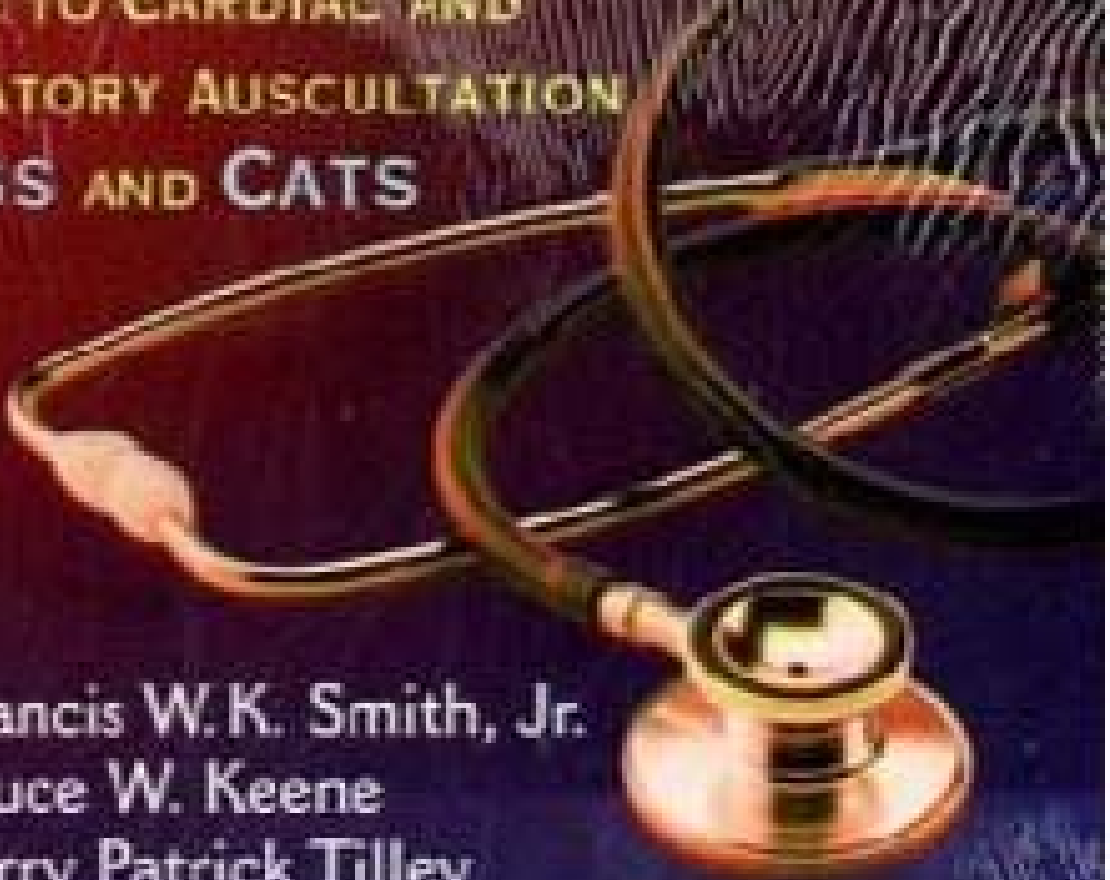
# Rapid Interpretation of Heart and Lung Sounds

## A Guide to Cardiac and Respiratory Auscultation in Dogs and Cats



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# RAPID INTERPRETATION OF HEART SOUNDS, MURMURS, AND ARRHYTHMIAS

## A GUIDE TO CARDIAC AUSCULTATION IN DOGS AND CATS

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# HEART SOUNDS

# TABLE OF ABBREVIATIONS

| | |
|---|---|
| $A_2$ | Aortic component of $S_2$ |
| AES | Aortic ejection sound |
| APC | Atrial premature complex |
| ASD | Atrial septal defect |
| AV | Atrioventricular |
| CCJ | Costochondral junction |
| cps | Cycles per second |
| DM | Diastolic murmur |
| ECG | Electrocardiogram |
| ES | Ejection sound |
| HOCM | Hypertrophic obstructive cardiomyopathy |
| ICS | Intercostal space |
| LBBB | Left bundle branch block |
| $M_1$ | First main component of $S_1$ |
| MSC | Mid-systolic click(s) |
| $P_2$ | Pulmonic component of $S_2$ |
| PAT | Paroxysmal atrial tachycardia |
| PDA | Patent ductus arteriosus |
| PES | Pulmonic ejection sound |
| PMI | Point of maximal intensity |
| PVT | Paroxysmal ventricular tachycardia |
| $S_1$ | First heart sound |
| $S_2$ | Second heart sound |
| $S_3$ | Third heart sound |
| $S_4$ | Fourth heart sound |
| SM | Systolic murmur |
| SS | Summation sound |
| $T_1$ | Second main component of $S_1$ |
| VD | Ventrodorsal |
| VPC | Ventricular premature complex |

## SOME PROPERTIES OF SOUND

Sound is produced and transmitted by the vibratory motion of matter. These vibrations initiate a series of compression waves that can travel through solid, liquid, or gaseous media. Sound waves have the following physical properties:

*INTENSITY.* The intensity of sound depends on the magnitude of air displacement of the sound wave. Intensity is determined by the energy of the sound source, the distance the sound travels, and the media it must go through to reach the receiver.

*FREQUENCY.* The frequency of sound is determined by the number of vibrations per second (cycles per second [cps]). The greater the cps, the higher the frequency.

*DURATION.* The duration of sound is controlled by the length of time of release of energy from the sound source. Cardiovascular sound consists of short-duration vibrations (heart sounds) and longer vibrations (heart murmurs).

The subjective characteristics of sound (perception of sound or hearing) are as follows:

*LOUDNESS.* The perceived loudness (or softness) of sound is determined by both intensity and frequency. A high-intensity sound in the ear's maximum auditory sensitivity range (1000 to 5000 cps) will be perceived as louder than an equally intense, lower frequency sound (i.e., 200 cps) because of the decrease of auditory sensitivity in the lower frequency range. Thus, intensity and loudness may move in the same direction when sounds are accentuated or diminished, but they are not synonymous.

*PITCH.* Pitch is the subjective perception of sound that is determined by frequency. The lower the frequency, the lower the pitch.

*TIMBRE.* Timbre is the distinguishing quality of a sound. The quality of most heart murmurs is determined by the frequency mix of the murmur.

## CARDIOVASCULAR SOUND

Most cardiovascular sound is produced within or about the heart and great vessels and is transmitted through liquid and solid media to the chest wall. During auscultation of the heart, we perceive air-transmitted sounds through the stethoscope. Cardiovascular sound can be divided into circumscribed sounds or transients (heart sounds) and longer combinations of vibrations (heart murmurs). Almost all clinically significant cardiovascular sound occurs in the frequency range of 20 to 500 cps (occasionally up to 1000 cps). The most commonly auscultated heart sounds in dogs and cats can be divided into (1) normal sounds ($S_1$ and $S_2$); (2) abnormal variations of $S_1$ and $S_2$; and (3) sounds that usually reflect cardiac disease such as $S_3$ and $S_4$ gallops, ejection sounds or clicks (ES), and mid-systolic or late-systolic clicks (MSC).

Auscultation of the heart is limited by two factors. First is the threshold sensitivity of the human ear. The normal adult can detect sound from 20 to 14,000 cps, but the most efficient range is 1000 to 5000 cps. Below 1000 cps, there is a progressive decrease in auditory sensitivity. Thus, an intense cardiovascular sound may be perceived as a soft sound and be difficult to hear. The relationship between the range of human hearing and cardiovascular sound is shown in Figure 1. The temporal relationships between heart sounds is also important. The ear normally will differentiate between a "slur" and two distinct sounds between 0.02 and 0.03 second. Therefore to appreciate a splitting of a heart sound or to differentiate between different heart sounds requires that the sounds be separated by at least this interval. Some heart sounds that are commonly auscultated in humans (i.e., physiologic splitting of $S_2$) are rarely heard in dogs and cats owing to the high heart rates in these species. Several intervals are depicted in Figure 2 and demonstrated on the tape. The second limiting factor is the stethoscope.

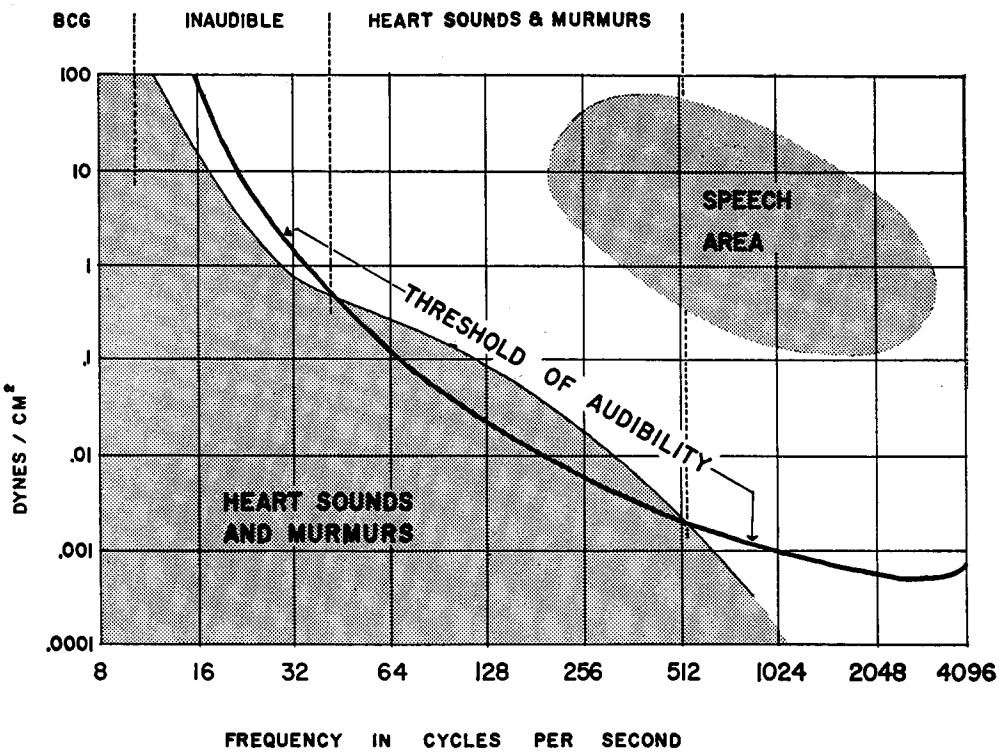


**Figure 1. Common frequency ranges of heart sounds and murmurs (From Butterworth, J.S., et al.: Cardiac Auscultation. New York, Grune & Stratton, 1960, p. 25.)**

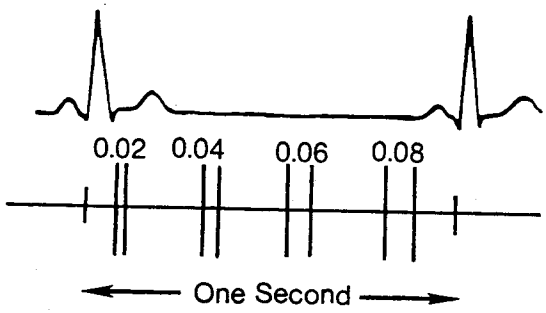


**Figure 2. Various intervals (in hundredths of a second).**

## THE STETHOSCOPE

The main components of the stethoscope are the bell, diaphragm, tubing, and ear pieces (Figure 3). The bell of an efficient stethoscope transmits both low-frequency (20 to 100 cps) and high-frequency (100 to 1000 cps) sounds. Larger bells cause less dampening of low-frequency sounds. Low-frequency components of a mixed-frequency sound "mask out" the high-frequency

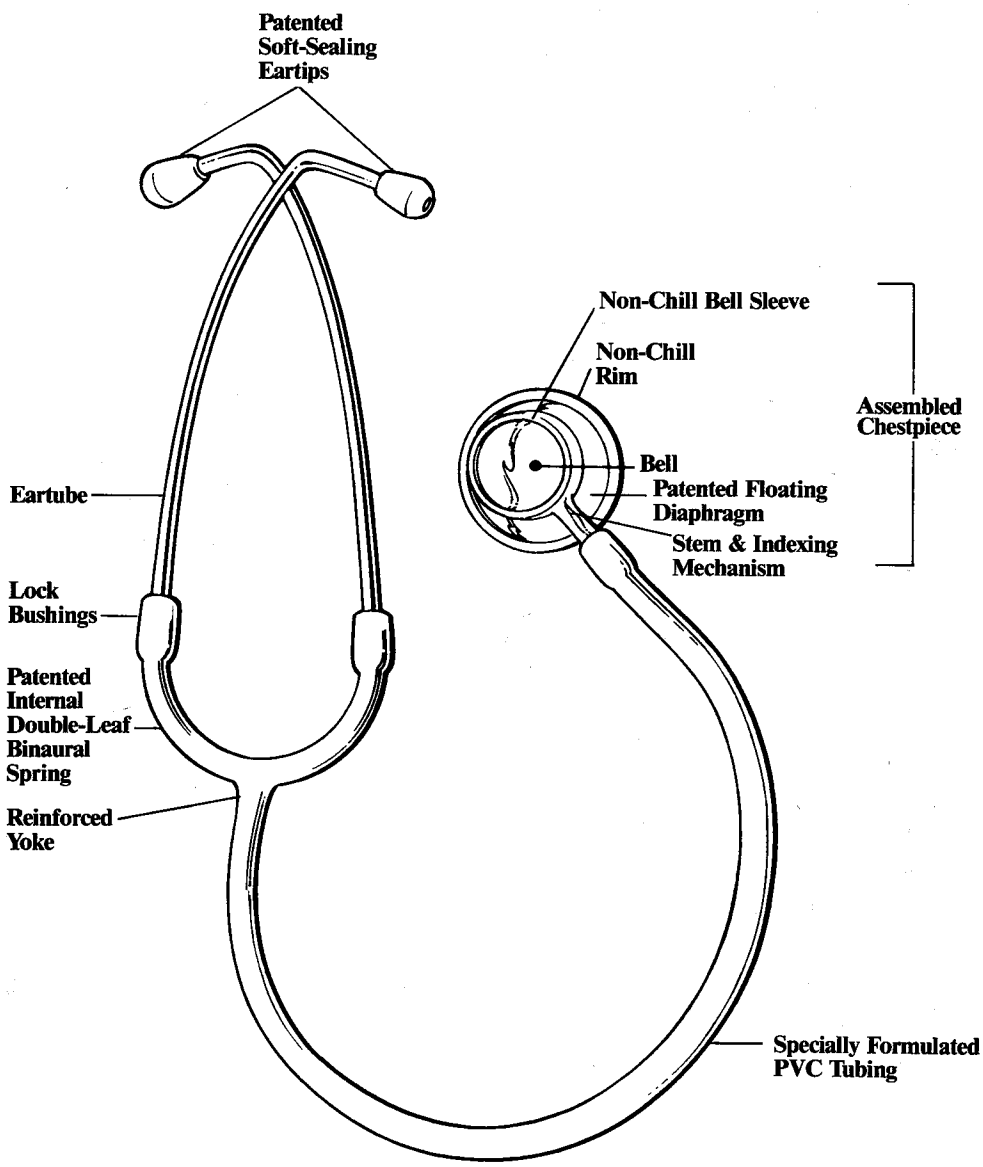


**Figure 3. Anatomy of a stethoscope with a combination chestpiece. (Courtesy of 3M Health Care, St. Paul, MN.)**

components. As a result, the high-frequency components may be perceived as faint or absent. The diaphragm of the stethoscope attenuates low frequencies (20 to 100 cps) and selectively transmits the high frequencies, which can then be auscultated. The diaphragm, by virtue of its larger size, transmits louder sounds than the bell. Most stethoscopes combine the bell and diaphragm into a dual-sided, combination-style chestpiece. A new stethoscope design by 3M, however, combines the bell and diaphragm into a single-sided chestpiece.

The Littman "Master" series stethoscopes from 3M Health Care use a patented single-sided chestpiece design that combines both the bell and the diaphragm modes onto the same side of the chestpiece (Figure 4). Simple fingertip pressure allows one to switch from low-frequency to high-frequency

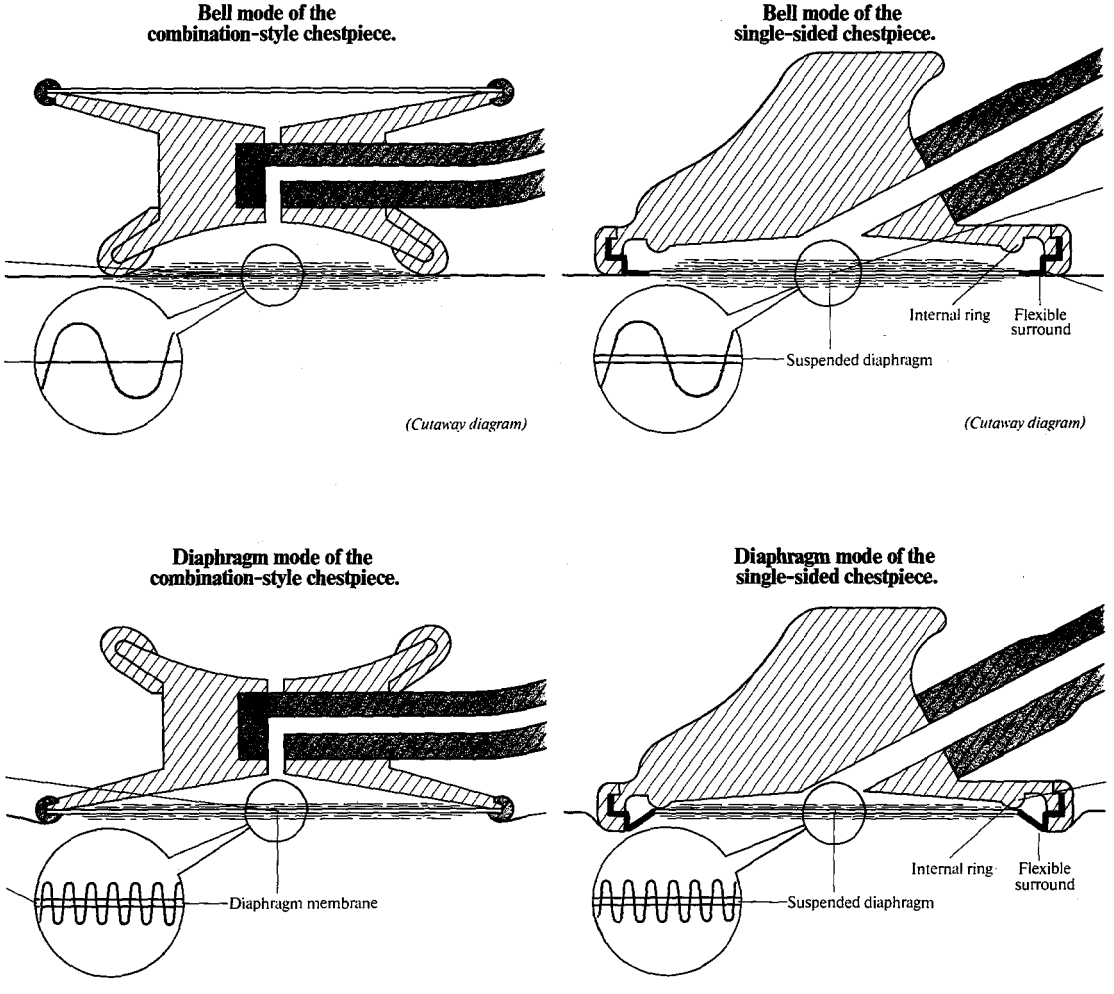


**Figure 4. The stethoscope on the left is the traditional design with separate bell and diaphragm (combination style chestpiece). The stethoscope on the right, the 3M Littmann Master Classic Stethoscope, is a newer design that incorporates the bell and diaphragm into a single head (single-sided chestpiece). Light pressure produces the effect of the bell, and increased pressure produces a diaphragm response. (Courtesy of 3M Health Care, St. Paul, MN.)**

sounds. The clinical benefit yielded by this new "Master" chestpiece design is that there is no interruption in sound as there is in a traditional two-sided stethoscope, resulting in added convenience and efficiency in auscultation.

The tubing should be flexible. Shorter tubing causes less attenuation of the heart sounds. A practical tubing length is approximately 14 to 18 inches. Thick-walled tubing is best for transmitting heart sounds and murmurs and reducing ambient noise. Ear tips should be comfortable and occlude the ear canal without entering the canal. Many types and sizes of ear tips are available. The most appropriate ear tips for any listener are best determined by trial and error. Ear tubes should angle forward to conform to the anatomy of the ear canal. This improves comfort and ear canal occlusion while auscultating. Electronic stethoscopes amplify background noise as well as heart sounds and therefore offer little practical benefit over standard stethoscopes.

## KEYS TO SUCCESSFUL AUSCULTATION

A properly fitted stethoscope is of great importance in the thorough examination of the heart. The ear pieces must be large enough to fit snugly without entering the ear canals. The chest piece should consist of a bell and a diaphragm in either a combination style or single-sided version. Appropriate use of the bell or diaphragm is critical to accurate auscultation. The bell is used to hear the lower frequency sounds and murmurs. The bell should be used with light pressure on the chest wall. Too much pressure on the bell tightens the skin and creates a diaphragm that filters out the lower frequencies.

Before starting the physical examination, the patient should be prepared for auscultation in a quiet room. Barking, purring, panting, and client conversation are major impediments to a successful examination. The patient should be standing. Close the mouth if the patient is panting. If the patient is excited and tachycardic, re-evaluate the heart after the patient relaxes. If the patient is still tachycardic and cardiac disease is suspected, vagal maneuvers can be attempted to slow the heart and permit more thorough and accurate auscultation. Care should be taken when auscultating not to confuse respiratory sounds, shivering, twitches, or rubbing of hair for heart sounds.

Develop a systematic approach to auscultation. Auscultate over all valve areas and over the carotid artery. Palpate a peripheral artery while auscultating the heart. The femoral artery is usually the easiest to palpate. The peripheral pulse occurs just after $S_1$ and helps distinguish $S_1$ from $S_2$. Pulse deficits suggest an arrhythmia, and pulse quality is an indirect indicator of cardiac output and blood pressure. Determine the point of maximal intensity, area of radiation, pitch, duration, quality, and timing of any murmurs. Note the effects of respiration on the rhythm, heart sounds, and murmurs. Concentrate on the overall and comparative loudness of the heart sounds. Conditions that uniformly alter the loudness of heart sounds are listed in Table 1.

TABLE 1. Conditions that Alter Loudness of All Heart Sounds

1. Increase loudness
   a. Thin-chested animals
   b. Vigorous ventricular contraction (hyperthyroidism, excitement)
2. Decrease loudness
   a. Obesity
   b. Pleural effusion
   c. Pericardial effusion
   d. Diaphragmatic or pericardial diaphragmatic hernia
   e. Pneumothorax
   f. Decreased ventricular contraction (hypothyroidism, dilated cardiomyopathy)

## PRINCIPAL AREAS OF CARDIAC AUSCULTATION

Frequent reference will be made to the four areas illustrated in Figure 5 and listed in Table 2. As you progress, you will find that additional areas are essential in cardiac auscultation.

TABLE 2. Principal Areas of Cardiac Auscultation

| | *DOG* | *CAT* |
|---|---|---|
| 1. Mitral Area | L 5 ICS at CCJ | L 5-6 ICS, $\frac{1}{4}$ VD distance from sternum |
| 2. Aortic Area | L 4 ICS above the CCJ | L 2-3 ICS just dorsal to pulmonic area |
| 3. Pulmonic Area | L 2–4 ICS at left sternal border | L 2–3 ICS, $\frac{1}{3}$–$\frac{1}{2}$ VD distance from sternum |
| 4. Tricuspid Area | R 3–5 ICS near CCJ | R 4–5 ICS, $\frac{1}{4}$ VD distance from sternum |

ICS = Intercostal space; CCJ = costochondral junction; VD = ventrodorsal.

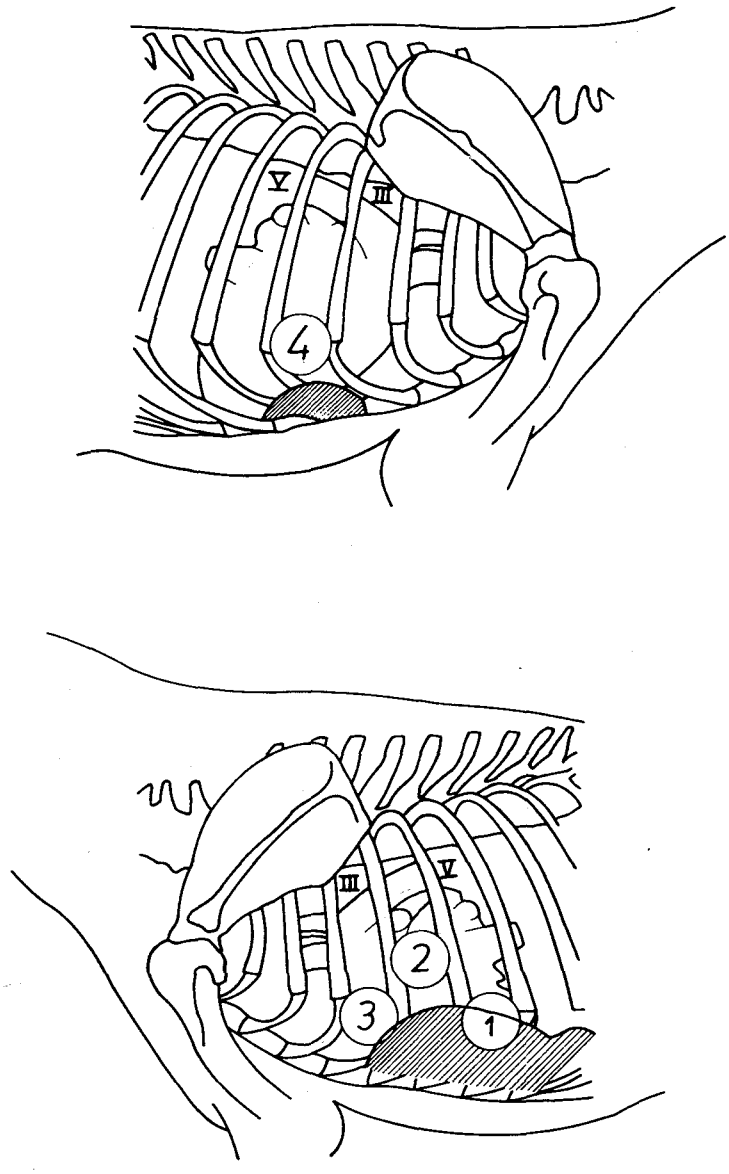


**Figure 5. Principal areas of cardiac auscultation in the dog. The valve relationships are the same in the cat. 1 = Mitral valve area; 2 = aortic valve area; 3 = pulmonic valve area; 4 = tricuspid valve area. Mitral, aortic, and pulmonic valves are auscultated on the left hemithorax. The tricuspid valve is auscultated on the right hemithorax. Shaded area = area of cardiac dullness. (From Detweiler, D.: Heart Sounds of the Dog. Ann. N.Y. Acad. Sci., 127:323-324, 1965.)**

## HEMODYNAMICS OF THE CARDIAC CYCLE

You will be better able to appreciate what you hear by understanding the hemodynamics of the cardiac cycle as illustrated in Figure 6. $M_1$, the first main component of the first heart sound ($S_1$) occurs at the time of or immediately

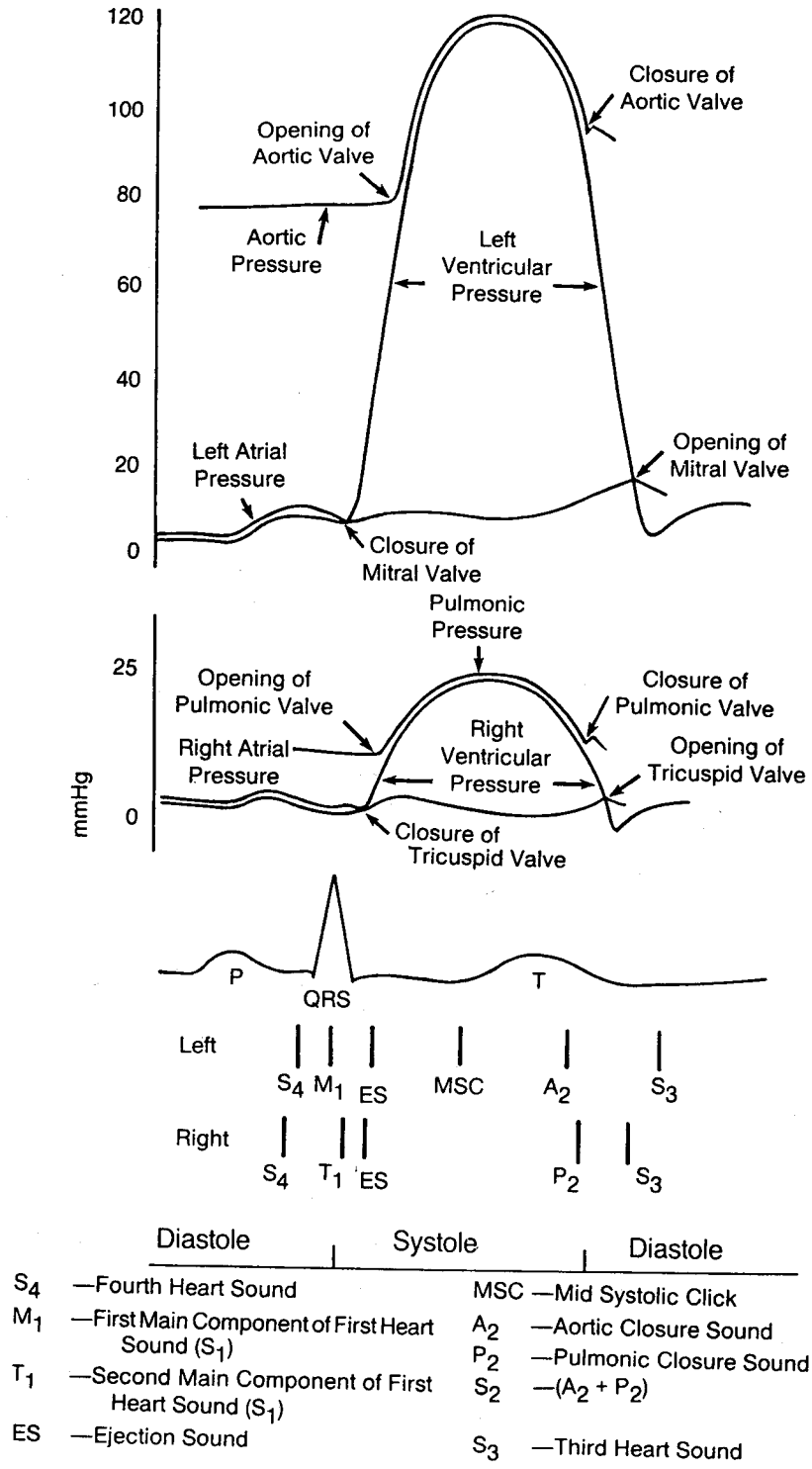


**Figure 6. Hemodynamics of the cardiac cycle.**

after mitral valve closure, in the early part of isovolumetric contraction of the left ventricle, before the onset of ventricular ejection. Thus $S_1$ is a useful signal of the onset of ventricular systole. The second heart sound ($S_2$) is normally composed of two components, aortic ($A_2$) and pulmonic ($P_2$), which occur at time of closure of the aortic and pulmonic valves, respectively. Thus $S_2$ signals the termination of ventricular systole.

## THE FIRST HEART SOUND ($S_1$)

We begin our study by listening in the mitral area to the characteristic "lub-dub" pattern of the normal heart sounds. $S_1$, which signals the onset of ventricular systole (see Figure 6), can be auscultated with both the bell and the diaphragm of the stethoscope. The two main components of $S_1$, however, have predominantly higher frequency vibrations, which are best delineated with the diaphragm. The first main component of $S_1$, the mitral component ($M_1$), is typically heard loudest at the cardiac apex. $M_1$ represents energy vibrations released from abrupt tension on the mitral valve apparatus at the time of or immediately after mitral valve closure at the beginning of systole. Audible variations in $S_1$ loudness are usually caused by alterations in $M_1$.

The second main component of $S_1$, the tricuspid component ($T_1$), is usually softer than $M_1$ and is loudest at the tricuspid area. Controversy exists surrounding the origin of $T_1$. Traditionally $T_1$ was thought to arise from the tricuspid valve at the time of valve closure. Recent evidence supports this view. Some investigators believe, however, that the audible $T_1$ is actually caused be events associated with the onset of ejection of blood into the aortic root in early ventricular systole. Physiologic splitting of $S_1$ is rarely appreciated in dogs and cats. It is heard occasionally as a normal variant on large and giant breeds of dogs. Pathologic splitting is caused by asynchronous closure of the atrioventricular valves caused by bundle-branch blocks and ventricular extrasystoles. When splitting is present, $S_1$ is heard as a split sound in the tricuspid area (Figure 7) and as a single sound in the aortic and mitral areas (Figures 8 and 9). A split $S_1$ must be differentiated from an $S_4$–$S_1$, $S_1$–ejection sound, and $S_1$–systolic click.

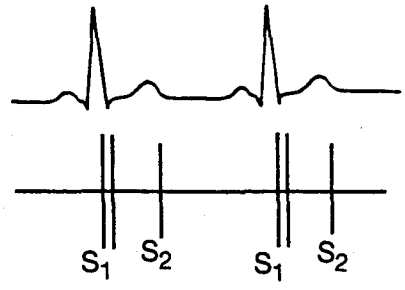


**Figure 7. Mitral and tricuspid components of $S_1$ (split sounds) and $S_2$ in the tricuspid area. Note $S_1$ is louder than $S_2$ in the tricuspid area. Rarely $S_1$ is heard as a split sound in the tricuspid valve area in giant breed dogs.**

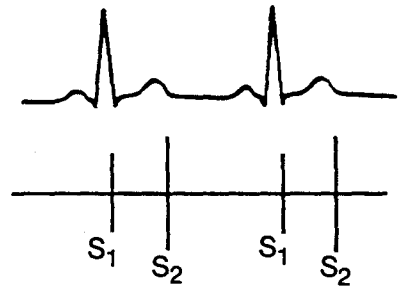


**Figure 8. $S_1$ and $S_2$ in the aortic area. Note that $S_2$ is louder than $S_1$ in the aortic area.**

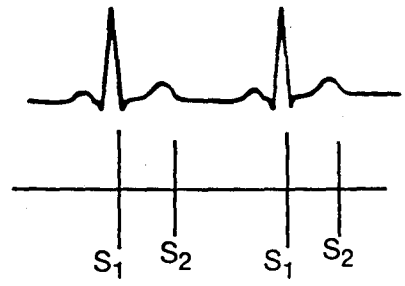


**Figure 9. $S_1$ and $S_2$ at the left apex. Note that $S_1$ is louder than $S_2$ at the left apex.**

Auscultation of the loudness of $S_1$ at the cardiac apex is useful in determining the presence of physiologic or anatomic abnormalities of the heart. The normal $S_1$ loudness must be compared with $S_2$ because many conditions alter the loudness of all heart sounds (see Table 1). Normal $S_1$ loudness may range from 0.5 to 2.0 times that of $S_2$ in the mitral area. In general, $S_1$ is also longer and lower pitched than $S_2$. The normal "lub-dub" pattern heard here usually consists of a single $S_1$ sound and a single $S_2$ sound (usually $A_2$).

Normal $S_1$ loudness occurs when there is a normal relationship between atrial and ventricular depolarization and contraction and a normal position of the mitral valve at onset of left ventricular contraction (normal P-R interval on the ECG), normal left ventricular function and isovolumetric contraction, and a normal mitral valve apparatus. Tables 3 and 4 list causes of an abnormally accentuated $S_1$ and an abnormally diminished $S_1$.

Varying $S_1$ loudness will occur with varying P-R intervals as in AV block or with marked variations in the R-R interval with atrial fibrillation, pronounced sinus arrhythmia, or atrial and ventricular premature complexes.

TABLE 3. Abnormally Accentuated $S_1$

1. Short P-R interval
2. Vigorous left ventricular contraction
   a. Pregnancy
   b. Hyperthyroidism
   c. Exercise
   d. Fever
   e. Anemia
   f. Systemic hypertension
   g. Inotropic agents
   h. Excitement or fear

TABLE 4. Abnormally Diminished $S_1$

1. Prolonged P-R interval (first-degree AV block)
2. Diminished left ventricular function
   a. Hypothyroidism
   b. Severe congestive heart failure—dilated cardiomyopathy
   c. Shock
3. Abnormal isovolumetric contraction
   a. Aortic regurgitation
   b. Mitral regurgitation
4. Heavy calcification or destruction of the mitral valve

## THE SECOND HEART SOUND ($S_2$)

We will now shift our auscultation from the cardiac apex to the base. $S_2$ is normally louder than $S_1$ at the base of the heart, is shorter, and is slightly higher pitched because of a greater number of higher frequency components. $S_2$ indicates the termination of systole, as diagrammed in Figure 6. $S_2$ is normally split into two components, aortic ($A_2$) and pulmonic ($P_2$), because of asynchronous closure of first the aortic and then the pulmonic valve. $A_2$ is normally heard first because systemic pressure is higher than pulmonary pressure and this forces the aortic valve closed earlier than the pulmonic valve. $A_2$ is usually audible over all the standard areas of cardiac auscultation and is best heard at the aortic and pulmonic areas. $P_2$ is normally softer than $A_2$ and is usually heard best at the pulmonic area. Therefore, separation of $S_2$ into its two components is best perceived at the heart base. At the mitral area, $S_2$ is usually heard as a single sound.

As mentioned previously, the ear normally will differentiate between a "slur" and two definite sounds between 0.02 and 0.03 second. Thus splitting of $S_2$ is audible only when $A_2$ and $P_2$ are separated by such an interval or greater. Splitting is best delineated with the diaphragm of the stethoscope. Auscultation of $S_2$ splitting should be performed during normal quiet respiration.

Normal or physiologic splitting of $S_2$ consists of widening of the $A_2$–$P_2$ interval during inspiration, with narrowing during expiration (Figure 10). These changes are primarily caused by respiratory mobility of $P_2$, with a lesser movement of $A_2$. During inspiration, there is a drop in intrathoracic pressure and fall in pulmonary vascular resistance and increased venous return to the right ventricle, pooling of blood in the lungs, and decreased venous return to the left ventricle. As a result, right ventricular ejection time is prolonged with a delay in $P_2$, and left ventricular ejection time is shortened with earlier occurrence of $A_2$. Physiologic splitting of $S_2$ usually cannot be detected in dogs and cats owing to the high heart rates and close proximity of $A_2$–$P_2$.

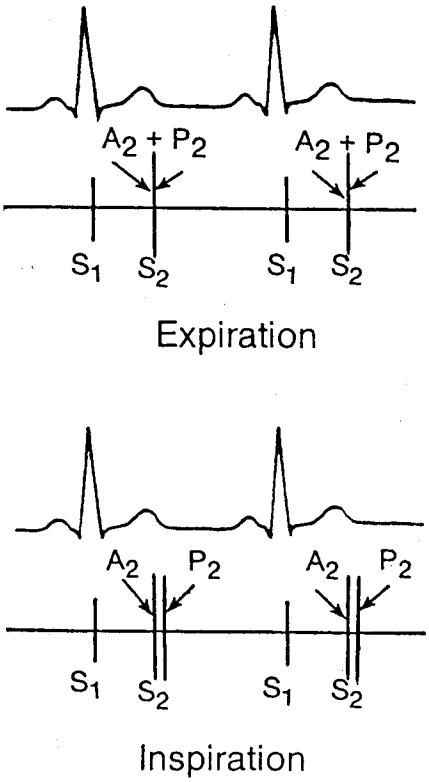


**Figure 10. Physiologic splitting of $S_2$.**

Abnormal splitting of $S_2$ usually indicates a cardiovascular abnormality. The types of abnormal splitting are (1) persistent splitting, (2) "fixed" splitting, and (3) paradoxical splitting.

In persistent splitting, the $A_2$–$P_2$ interval is wider than normal throughout the respiratory cycle. There is normal widening during inspiration and narrowing with expiration but without expiratory fusion of $S_2$ (Figure 11). Causes of persistent splitting are listed in Table 5.

TABLE 5. Persistent Splitting of $S_2$

1. Pulmonic stenosis
2. Significant mitral regurgitation
3. Complete right bundle branch block
4. Ventricular septal defect
5. Heartworm disease

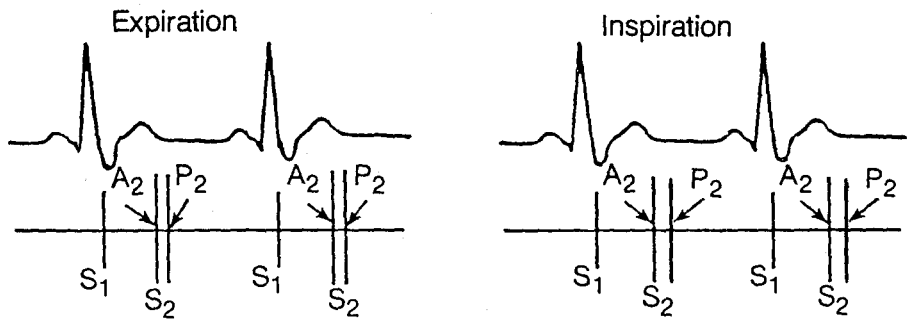


**Figure 11. Persistent splitting of $S_2$ in a dog with heartworm disease.**

In fixed splitting, the $A_2$–$P_2$ interval is also wider than normal throughout the respiratory cycle but with minimal respiratory variation (usually less than 0.010 to 0.015 second). The $S_2$ split sounds "fixed" during auscultation, with auditory inspiratory and expiratory splitting present (Figure 12). Table 6 lists some causes of "fixed" splitting.

TABLE 6. "Fixed" Splitting of $S_2$

1. Severe valvular pulmonic stenosis
2. Mitral regurgitation with significant left ventricular dysfunction
3. Right bundle branch block with significant left ventricular dysfunction
4. Atrial septal defect

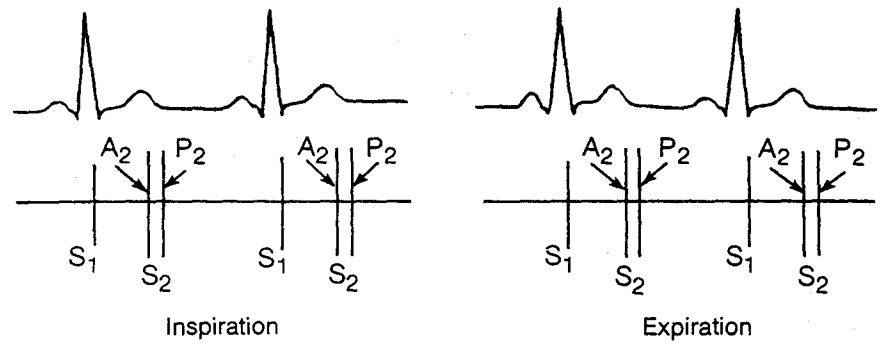


**Figure 12. Fixed splitting of $S_2$ in a patient with an atrial septal defect.**

In paradoxical splitting, when aortic valve closure is significantly delayed with respect to pulmonic valve, the aortic valve may close after the pulmonic valve, reversing the normal sequence. $P_2$ now precedes $A_2$. The $S_2$ split increases during expiration and decreases with inspiration, resulting in paradoxical splitting (Figure 13). Conditions that may be associated with paradoxical splitting are listed in Table 7.

TABLE 7. Paradoxical Splitting of $S_2$

1. Left bundle branch block
2. Right ventricular pacemaker
3. Patent ductus arteriosus
4. Aortic stenosis
5. Significant aortic regurgitation
6. Significant systemic hypertension

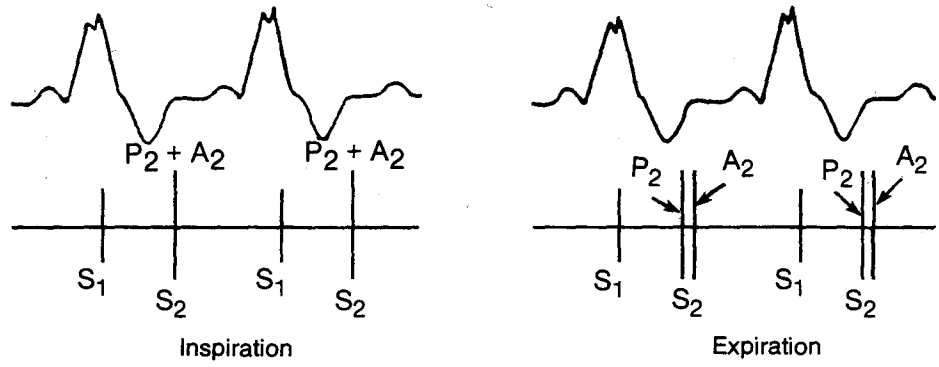


**Figure 13. Paradoxical splitting of $S_2$ in a patient with left bundle branch block (LBBB).**

The loudness of $S_2$ is determined by the sum of $A_2$ and $P_2$. An abnormally accentuated $S_2$ may be caused by abnormal augmentation of either $S_2$ component. Causes of abnormal accentuation of $A_2$ and $P_2$ are listed in Table 8. When $P_2$ is abnormally accentuated, it is significantly louder than $A_2$ in the pulmonic area. Conditions that may be associated with abnormal diminution of $S_2$ are listed in Table 9.

TABLE 8. Abnormally Accentuated $S_2$

1. Abnormally accentuated $A_2$
   a. Systemic hypertension
   b. Aortic dilatation or aneurysm of ascending aorta
   c. Valvular aortic stenosis (noncalcified valve)
2. Abnormally accentuated $P_2$
   a. Pulmonary hypertension secondary to congestive heart failure
   b. Significant mitral valve disease
   c. Congenital left-to-right shunts
      (1) Patent ductus arteriosus
      (2) Ventricular septal defect
      (3) Atrial septal defect
   d. Primary pulmonary hypertension
   e. Pulmonary embolism
   f. Idiopathic dilatation of the pulmonary artery
   g. Mild valvular pulmonic stenosis

TABLE 9. Abnormally Diminished $S_2$

1. Total diminution of $S_2$ (markedly decreased ventricular function)
   a. Hypothyroidism
   b. Shock
   c. Dilated cardiomyopathy
2. Diminished $A_2$
   a. Significant calcific valvular aortic stenosis
   b. Marked aortic regurgitation
3. Diminished $P_2$
   a. Significant pulmonic stenosis of any cause

$S_2$ may not be audible in the presence of certain arrhythmias. Depending on the timing and nature of the ectopic beat, there may not be adequate ventricular filling to cause opening of the semilunar valves.

## THE THIRD HEART SOUND ($S_3$)

Blood accelerates into the ventricle during the rapid filling phase in early diastole. The impingement of the inflowing blood on the apical region of the ventricle results in sudden outward acceleration that ends abruptly at maximal distention, with resultant release of energy vibrations. If energy release is great enough, an audible vibration ($S_3$) will be auscultated (see Figure 6). $S_3$ is usually a left ventricular sound and is heard best at the left cardiac apex. Occasionally a right ventricular $S_3$ occurs and is best auscultated in the tricuspid area. See Table 10 for causes of $S_3$.

TABLE 10. Causes of $S_3$ (Protodiastolic Gallop)

1. High cardiac output
   a. Large left-to-right shunts (ventricular septal defect, patent ductus arteriosus)
   b. Anemia
   c. Thyrotoxicosis
2. Excessively rapid filling of either ventricle
   a. Mitral regurgitation
   b. Tricuspid regurgitation
   c. Aortic regurgitation
3. Reduced diastolic filling superimposed on abnormal residual ventricular volume or compliance (overt or latent congestive heart failure of any cause)

$S_3$ is a low-frequency sound and therefore is heard best with the bell of the stethoscope lightly applied to the chest wall (Figure 14). The sound may be accentuated by maneuvers that increase blood flow to the heart (exercise). Reduction of venous return by prolonged rest will cause a reduction in loudness or a disappearance of an $S_3$. $S_3$ will also show characteristic respiratory variation in loudness, with the left ventricular $S_3$ accentuated during expiration and the occasional right ventricular $S_3$ augmented during inspiration. $S_3$ is considered pathologic in dogs and cats. It is called a protodiastolic or ventricular gallop.

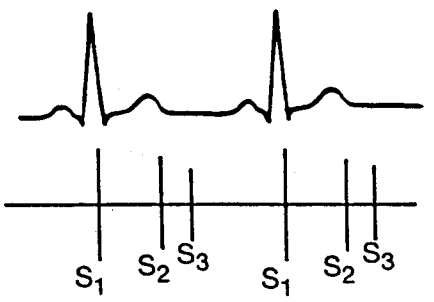


**Figure 14. $S_3$ in a Doberman Pinscher with dilated cardiomyopathy.**

## THE FOURTH HEART SOUND ($S_4$)

The audible component of $S_4$ occurs in presystole with active ventricular filling (see Figure 6). It follows atrial contraction, occurring just before $S_1$. The audible or "clinical" component of $S_4$ probably has a similar genesis as that of $S_3$ but occurs with the inflow of blood after atrial contraction in patients with abnormal ventricular compliance. $S_4$ is also called an atrial gallop or presystolic gallop. $S_4$ may be a right-sided or left-sided sound. A right-sided $S_4$ (from the right ventricle) is heard best in the tricuspid area and increases in loudness during inspiration. A left-sided $S_4$ (from the left ventricle) is auscultated best at the left cardiac apex and is augmented during expiration. A presystolic ($S_4$) gallop is considered pathologic in the dog and cat. Cardiac conditions that may be associated with a right-sided or left-sided $S_4$ are listed in Table 11.

TABLE 11. Conditions Associated with $S_4$

1. Right-sided $S_4$
   a. Pulmonary hypertension of any cause
   b. Cardiomyopathy
   c. Significant valvular pulmonic stenosis
2. Left-sided $S_4$
   a. Systemic hypertension and hypertensive heart disease
   b. Significant valvular and subvalvular aortic stenosis
   c. Cardiomyopathy
   d. Acute mitral regurgitation (damaged chordae tendineae)
   e. Chronic mitral regurgitation
3. Isolated $S_4$ (no associated $S_1$ or $S_2$)
   a. Second-degree AV block
   b. Complete AV block

$S_4$ is the lowest pitched heart sound (Figure 15). It is heard best with the bell of the stethoscope lightly applied to the chest wall. As with an $S_3$, the sound may be accentuated by increasing blood flow to the heart with exercise. Maneuvers reducing venous return will also diminish an $S_4$ or cause it to disappear.

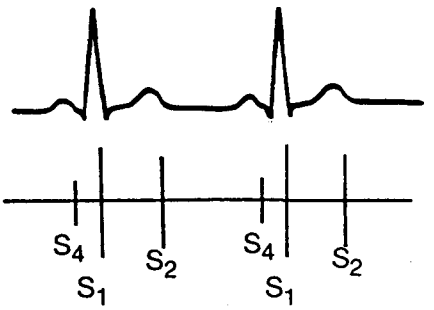


**Figure 15. $S_4$ in a cat with hypertrophic cardiomyopathy.**

## QUADRUPLE RHYTHM AND SUMMATION SOUND OR GALLOP

At times all four heart sounds may be heard, forming a quadruple rhythm (Figure 16). When both $S_3$ and $S_4$ are present and the heart rate accelerates, they may come together in diastole to be heard as a single sound known as a summation sound (SS) or summation gallop (Figure 17). This occurs commonly in cats with heart disease, owing to their high heart rates.

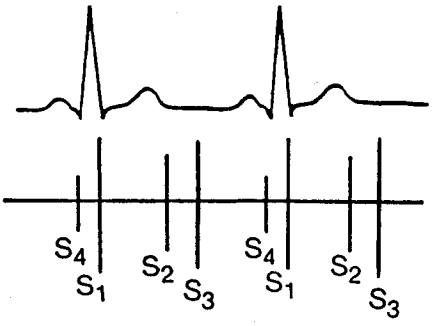


**Figure 16. A quadruple rhythm formed by $S_1$, $S_2$, $S_3$, and $S_4$.**

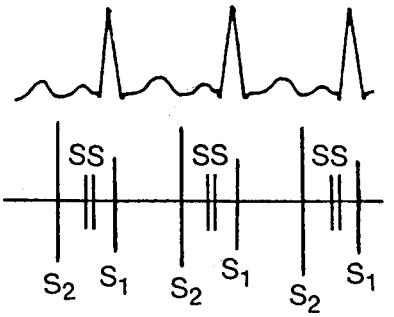


**Figure 17. A summation sound (SS) formed by $S_3$ plus $S_4$ in a cat with dilated cardiomyopathy.**

## EJECTION SOUNDS OR CLICKS

Ejection sounds or clicks (ES) are discrete, high-frequency sounds following the first main component of $S_1$ ($M_1$) and occurring at the time of onset of ventricular ejection (Figure 18). ES may arise from either the aortic or pulmonic circulation. The terms ejection sound and ejection click are frequently used interchangeably, depending on the quality of the sound. When the sound is very high pitched and "clicky," it is called an ejection click. When the "clicky" quality is absent, it is often referred to as an ejection sound. Ejection sounds or clicks are uncommon in dogs and cats.

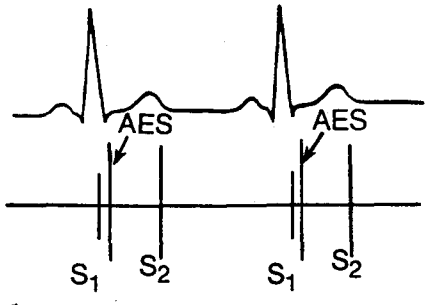


**Figure 18. Aortic ejection sounds (AES) in a patient with aortic valve stenosis.**

Aortic ejection sounds (AES) are heard best at the left heart base. AES appear to have two causes. The first type of AES results from energy release and vibrations with onset of ejection of blood from the left ventricle into the aorta and may represent accentuation of the normal second main component of $S_1$. This AES may occur with increased or forceful flow into the aorta or with systemic hypertension and is associated with aortic root dilatation. The second type of AES occurs in valvular aortic stenosis at the moment of termination of the opening movements of the abnormal aortic valve.

Because AES are high-pitched sounds, they are heard best with the diaphragm of the stethoscope. Recognition of an AES during auscultation depends on the relative loudness of $S_1$ and AES and the interval separating the sounds. The sound combination should be differentiated from an $S_4$–$S_1$ or split $S_1$ combination. The high pitch of ES helps differentiate them from the low-pitched $S_4$. Table 12 lists clinical conditions that may be accompanied by an AES.

TABLE 12. Aortic Ejection Sounds

1. Associated with forceful left ventricular ejection
   a. Hyperthyroidism
   b. Exercise
   c. Anemia
   d. Other high-output states
2. Dilatation of the ascending aorta with or without systemic hypertension
3. Valvular aortic stenosis

Pulmonic ejection sounds (PES) are auscultated best in the pulmonic area and along the left sternal border. PES follow $M_1$ (Figure 19). PES may be associated with valvular pulmonic stenosis or may arise from the pulmonary artery with or without pulmonary hypertension. PES occur with the onset of ejection of blood from the right ventricle into the pulmonary artery. The PES with valvular pulmonic stenosis typically occur earlier and have a characteristic respiratory variation in loudness (diminished during inspiration and accentuated during expiration) not seen with the pulmonary artery root ES. PES are also heard best with the diaphragm of the stethoscope. Cardiac conditions that may be associated with a PES are listed in Table 13.

TABLE 13. Pulmonic Ejection Sounds

1. Dilated main pulmonary artery with pulmonary hypertension
   a. Atrial septal defect
   b. Recurrent pulmonary emboli
   c. Primary pulmonary hypertension
2. Dilated main pulmonary artery without pulmonary hypertension
   a. Idiopathic dilatation of pulmonary artery
   b. Atrial septal defect without hypertension
3. Valvular pulmonic stenosis

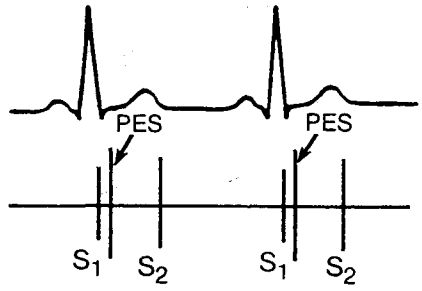


**Figure 19. Pulmonic ejection sound (PES) in a patient with pulmonary valve stenosis.**

## MID-SYSTOLIC CLICK OR CLICKS

Mid-systolic clicks (MSC) are discrete, high-frequency sounds that usually occur during mid-ventricular or late-ventricular systole (Figure 20). These sounds are best auscultated with the diaphragm of the stethoscope over the mitral and tricuspid valves. MSC may be present alone, may initiate or occur during mid-systolic to late-systolic murmurs, or may be obscured by a holosystolic murmur. Lability of timing and intensity characterizes the MSC, which at any given time may be absent, single, or multiple and may be either mid-systolic or late-systolic in occurrence. In humans, most MSC are attributed to sudden tension of redundant chordae tendineae or leaflets of the mitral valve when abnormal mitral valve prolapse occurs during ventricular systole. They are heard occasionally in dogs with myxomatous degeneration of the mitral valve. Systolic clicks have also been noted as incidental findings in dogs without apparent cardiac disease.

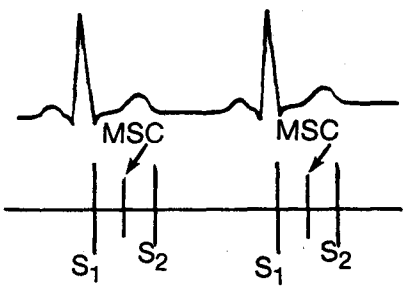


**Figure 20. Mid-systolic clicks (MSC) in an animal with mitral valve prolapse.**

## ANSWERS TO PRETEST 1

1. Right bundle branch block
2. Atrial septal defect
3. First heart sound
4. Fourth heart sound
5. False
6. True
7. True
8. False
9. False
10. True

# POST-TEST 1

## PART A

*DIRECTIONS.* Part A consists of 10 questions. After determining the correct answers, fill in the appropriate blanks.

1. Left bundle-branch block can cause paradoxical splitting of $S_2$. True or False _____.
2. Ejection sounds are common in cats and dogs. True or False _____.
3. $S_2$ is louder in patients with severe pulmonic stenosis. True or False _____.
4. The left ventricular $S_3$ is heard best at the left apex. True or False _____.
5. During inspiration, blood flow to the right ventricle is increased. True or False _____.
6. Persistent splitting of $S_2$ is seen with canine heartworm disease. True or False _____.
7. $S_3$ is a lower pitched heart sound than $S_2$. True or False _____.
8. Summation gallops are uncommon in cats. True or False _____.
9. $S_3$ is considered normal in puppies and kittens. True or False _____.
10. $S_3$ is a low-frequency sound that is heard best with the bell of the stethoscope. True or False _____.

## PART B

*DIRECTIONS.* Part B consists of 10 unknowns presented on your cassette tape, side 1. After determining the correct answers, fill in the appropriate blanks. Pay close attention to the location and timing of the heart sounds. Because you are not examining the patient, the location and, where appropriate, the timing are provided.

1. Apex. _____.
2. Aortic area. Identify the early systolic sound. _____.
3. Apex. Identify the diastolic sound. If the rate were lower, two diastolic sounds would be heard. _____.
4. Pulmonic area. _____.
5. Apex. Identify the sound following $S_2$. _____.
6. Apex. Identify the mid-systolic sounds. _____.
7. Pulmonic area. Identify the early systolic sound. _____.
8. Apex. Identify the sound preceding $S_1$. _____.
9. Pulmonic area. This Great Dane has a normal ECG. _____.
10. Pulmonic area. _____.

# ANSWERS TO POST-TEST 1

## PART A

1. True
2. False
3. False
4. True
5. True
6. True
7. True
8. False
9. False
10. True

## PART B

1. Quadruple rhythm
2. Aortic ejection sound
3. Summation sound
4. "Fixed" splitting of $S_2$
5. Third heart sound ($S_3$)
6. Mid-systolic clicks
7. Pulmonic ejection sound
8. Fourth heart sound ($S_4$)
9. Physiologic splitting of $S_2$
10. Persistent splitting of $S_2$

# MURMURS

## OBJECTIVES (SECTION 2)

Upon completion of this program, you should be able to:

1. List the steps to be employed to evaluate heart murmurs properly.
2. Explain the grading of murmurs.
3. Explain, on the basis of frequency (cps) and timing, the various types of systolic and diastolic murmurs.
4. State the characteristics of the murmur of aortic stenosis.
5. List the auscultatory findings associated with ventricular septal defects.
6. List criteria that are useful in differentiating innocent or physiologic from pathologic murmurs.
7. Recognize the auscultatory findings of mitral regurgitation.
8. Explain the origin of the systolic murmur of atrial septal defect.
9. State the effect of respiration on the murmur of tricuspid regurgitation.
10. Recognize the murmur of patent ductus arteriosus.

## PRETEST 2

*DIRECTIONS.* This pretest consists of 10 questions. After determining the correct answers, fill in the appropriate blanks.

1. A palpable sensation, known as a thrill, often accompanies a grade _____ murmur.
2. The point of maximal intensity of the murmur of a ventricular septal defect is _____.
3. Crescendo-descrescendo murmurs are characteristic of _____ and _____.
4. The intensity of physiologic flow murmurs is often grade III or higher. True or False _____.
5. The murmur of aortic stenosis may radiate up the carotids to the top of the head. True or False _____.
6. The murmur in atrial septal defect is due to the turbulence of blood flow across the defect. True or False _____.
7. A low-pitched $S_3$ may be heard in association with mitral regurgitation. True or False _____.
8. Is the murmur of tricuspid regurgitation accentuated or diminished on inspiration? _____.
9. The murmur in ventricular septal defect is caused by blood flow across the defect. True or False _____.
10. The systolic murmur of aortic stenosis and the diastolic murmur of aortic regurgitation form a to-and-fro murmur. True or False _____.

Answers on page 52.

## EVALUATION OF HEART MURMURS

To evaluate heart murmurs, the following must be considered:

*TIMING, CONFIGURATION, AND DURATION.* Is the murmur occurring in systole or diastole, or is it a continuous murmur? In addition to gross timing, it is also important to know whether the murmur is occurring in early, mid, or late systole or diastole, and what its configuration and duration are. Terms used to describe the configuration of murmurs include plateau, crescendo-decrescendo or diamond-shaped, and decrescendo. These are depicted in Figure 21.

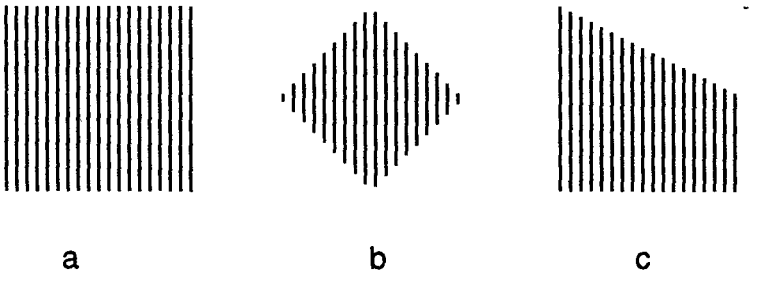


**Figure 21. Common configurations of cardiac murmurs (*a*, plateau; *b*, crescendo-decrescendo; *c*, decrescendo).**

*PITCH AND TIMBRE.* The pitch and quality of heart murmurs are determined by the frequency range and frequency mix of the murmurs. When the frequency range is 60 to 100 cps, the murmur is low pitched and rumbling. With a frequency range of 100 to 250 cps, the murmur is of medium pitch, rough, or harsh. With a frequency greater than 300 cps, the murmur is high pitched or blowing.

*POINT OF MAXIMAL LOUDNESS AND RADIATION.* The next step is to locate the point of maximal loudness of the murmur and to study the radiation or transmission of the murmur.

*RESPIRATION.* Does the murmur vary with respiration and if so how?

*GRADING LOUDNESS.* The loudness of systolic murmurs is grade form I to VI.

Grade I is a very faint murmur, requiring concentration to be heard.
Grade II is a faint murmur but is heard immediately on placing the stethoscope on the chest.
Grade III is an intermediate murmur, louder than grade II. Most hemodynamically significant murmurs are at least grade III.

Grade IV is a loud murmur, frequently associated with a palpable sensation, known as a thrill.

Grade V is a very loud murmur, still requiring at least the edge of the stethoscope to remain in contact with the chest to be audible.

Grade VI is a murmur audible with the stethoscope just breaking contact with the chest.

Knowledge of the timing and location of murmurs allows one to establish a differential diagnosis rapidly (Figure 22). This differential diagnosis can often be narrowed further by considering the breed. This is especially true for congenital defects (Table 14). Systolic murmurs developing in mature small breed dogs are typically caused by mitral and or tricuspid valve degeneration. Acquired systolic murmurs in large breed dogs are usually caused by dilated cardiomyopathy.

TABLE 14. Breed Predilections for Cardiac Disorders

| *DISORDER* | *BREED PREDILECTION* |
|---|---|
| Aortic stenosis | Boxer, German Shepherd, Golden Retriever, Newfoundland, Rottweiler |
| Atrial septal defect | Samoyed |
| Mitral insufficiency | Chihuahua, English Bulldog, Great Dane |
| Patent ductus arteriosus | Cocker Spaniel, Collie, German Shepherd, Irish Setter, Pomeranian, Poodle, Shetland Sheepdog |
| Pulmonic stenosis | Beagle, Chihuahua, English Bulldog, Schnauzer, terriers |
| Tetralogy of Fallot | Keeshond, English Bulldog |
| Tricuspid insufficiency | Great Dane, Weimaraner |
| Ventricular septal defect | English Bulldog |
| Degenerative AV valve disease | Small breed dogs |
| Dilated cardiomyopathy | Large and giant breed dogs |
| Hypertrophic cardiomyopathy | Persian cats |

Cardiac murmurs can be attributed to three main factors: (1) high flow through normal or abnormal valves; (2) forward flow through a stenosed or irregular valve, ventricular outflow tract obstruction, or into a dilated great vessel or ventricle; (3) backward or regurgitant flow through an incompetent valve, ventricular septal defect, or patent ductus arteriosus. Various combinations of these factors may be present and determine the timing, configuration, duration, pitch timbre, and loudness of the murmur.

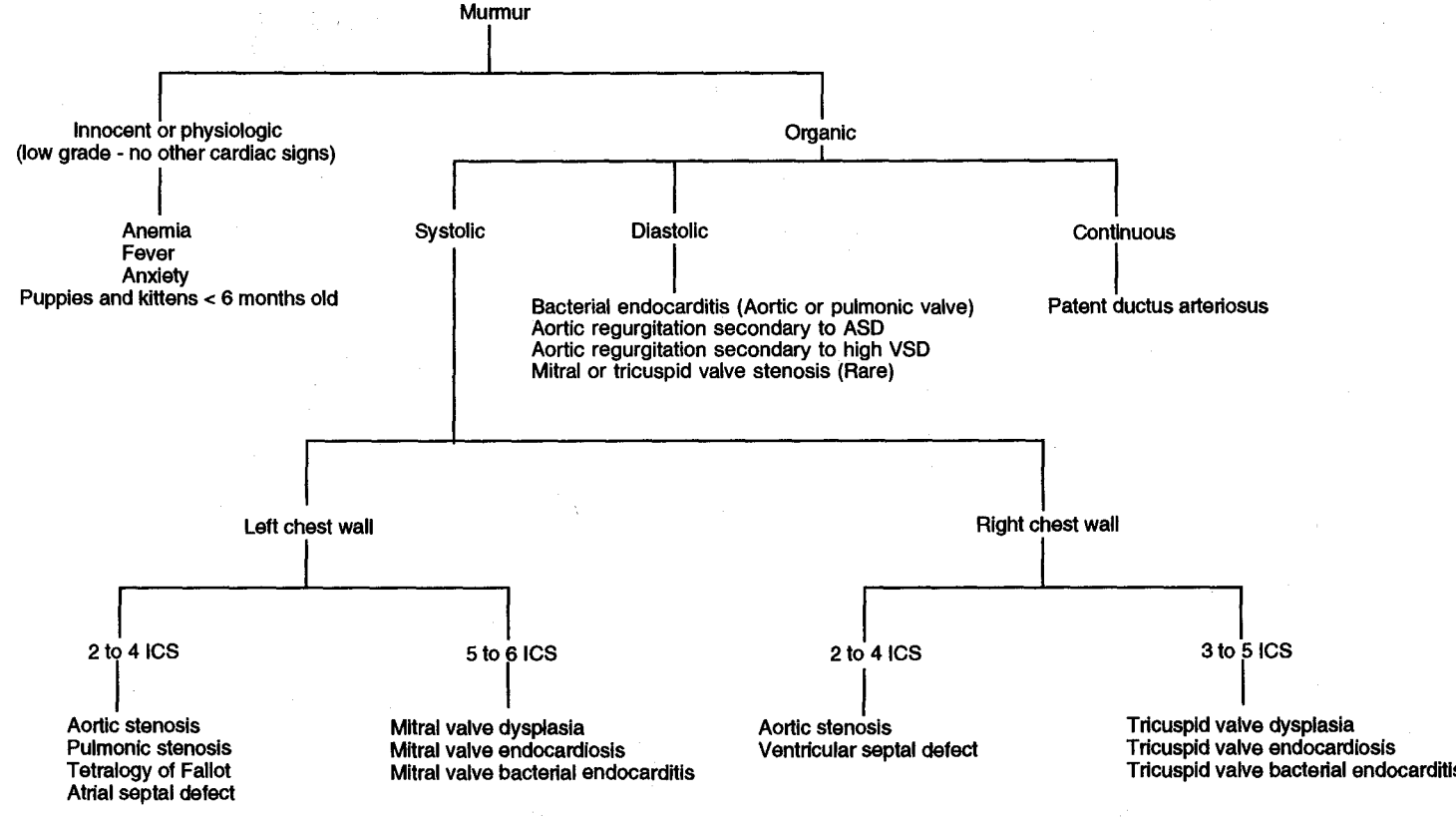


**Figure 22. Differential diagnosis of cardiac disease based on the timing and location of murmurs. (Adapted from Allen, D.G.: Murmurs and abnormal heart sounds. In Small Animal Cardiopulmonary Medicine. Edited by Allen, D.G., Kruth, S.A. Philadelphia, BC Decker, 1988, p. 13.**

## SYSTOLIC MURMURS

Systolic murmurs are auscultated and recorded between $S_1$ and $S_2$, during the phases of left or right ventricular systole. Systolic murmurs may be divided into three main groups:

Ejection systolic murmurs
Holosystolic or regurgitant murmurs
Late systolic murmurs

## EJECTION SYSTOLIC MURMURS

Ejection systolic murmurs start shortly after (not with) $S_1$, increase to a peak loudness in early systole to mid-systole, and then decrease in loudness and terminate before the respective component of $S_2$ ($A_2$, $P_2$). These murmurs are typically diamond-shaped or crescendo-decrescendo in contour. Causes of ejection systolic murmurs are listed in Table 15.

TABLE 15. Causes of Ejection Systolic Murmurs

1. Left ventricular outflow obstruction
   a. Supravalvular aortic stenosis
   b. Valvular aortic stenosis
   c. Discrete subaortic stenosis
   d. Hypertrophic cardiomyopathy
2. Right ventricular outflow tract obstruction
   a. Valvular pulmonic stenosis
   b. Infundibular pulmonic stenosis
   c. Tetralogy of Fallot
3. Hyperkinetic or high-flow states
   a. Congenital left-to-right shunt (i.e., ASD)
   b. Anemia
   c. Thyrotoxicosis
4. Functional (or innocent) systolic murmurs
5. Miscellaneous
   a. Dilatation of aorta or pulmonary artery distal to semilunar valves
   b. Degenerative changes in aortic valve without significant stenosis

ASD = atrial septal defect.

Significant lesions in categories 1 and 2 of Table 15 are typically associated with the prolonged, louder (grades III to VI) systolic murmurs with a delayed peak. Such murmurs are best initially auscultated with the diaphragm of the stethoscope for clearest delineation of the main components of $S_1$ and $S_2$ and the dominant medium (with some high) frequencies of the systolic murmur.

Murmurs occurring with minimal or absent left or right outflow tract obstruction, or categories 3 to 5, are typically shorter in duration, softer (grades I to II), and with an early peak in loudness in the first third to half of systole. These murmurs have dominant medium (with some low) frequencies and are best initially auscultated with the bell of the stethoscope. Several characteristic ejection systolic murmurs will now be studied.



Figure 22. Differential diagnosis of cardiac disease based on the timing and location (Adapted from Allen, D.G.: Murmurs and abnormal heart sounds. In Small Animal Cardiopulmonary Medicine. Edited by Allen, D.G., Kruth, S.A. Philadelphia, BC Decker, 1988, p. 13.

*Valvular aortic stenosis*, when hemodynamically significant, reflects pathologic narrowing of the aortic valve to less than 50% of its normal area. This is a very rare congenital defect in dogs and will not be discussed further.

The systolic murmur of *subaortic stenosis* results from abnormal blood flow across a left ventricular outflow obstruction below the aortic valve. This is the most common form of aortic stenosis in the dog, constituting approximately 90% of all cases. The murmur is usually loudest in the aortic area and frequently radiates to the right thorax (thoracic inlet to the third intercostal space). The murmur may radiate up the carotids and rarely to the calvarium. It is also a harsh or rough, medium-pitched to high-pitched murmur, frequently of long duration. As with valvular aortic stenosis, as a general rule, the more severe the obstruction, the longer and louder is the murmur. Unlike valvular aortic stenosis, the left ventricular outflow obstruction may be labile rather than fixed. In such patients, the loudness and duration of the murmur may vary significantly from examination to examination, with changes determined by the hemodynamic state of the patient.

Similar auscultatory findings may be present in cats with *hypertrophic cardiomyopathy* and *aortic outflow obstruction* (Figure 23). It should be emphasized that all cats with hypertrophic cardiomyopathy do not have aortic outflow obstruction. Cats with hypertrophic cardiomyopathy may also have regurgitant murmurs in the mitral valve area caused by distortion of the mitral valve annulus.

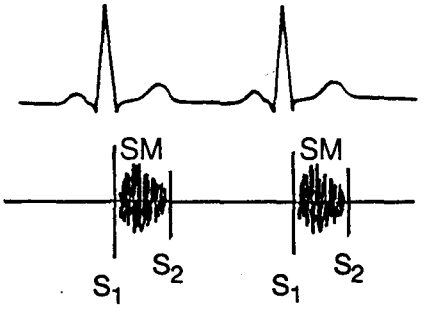


**Figure 23. Ejection systolic murmur (SM) in a cat with hypertrophic cardiomyopathy.**

Auscultatory clues in significant subaortic stenosis are as follows:

1. Harsh, medium-pitched, loud (grade III or greater), long ejection systolic murmur with crescendo-decrescendo pattern (Figure 24).
2. Murmur typically loudest in the aortic area and radiating to the area of the right thoracic inlet; murmur may radiate up the carotids and rarely to the calvarium.
3. Normal to accentuated $S_1$.
4. Rarely hear aortic ejection sound (Figure 25).
5. May hear paradoxical splitting of $S_2$.
6. Frequent variation in loudness and duration of the systolic murmur from examination to examination (particularly with labile obstruction).
7. Murmur not always auscultable from birth; usually develops within the first year of life.

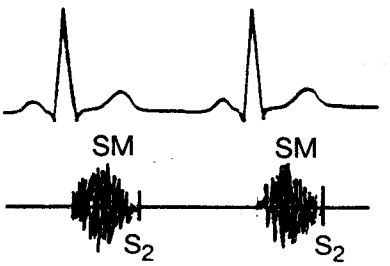


**Figure 24. Crescendo-decrescendo systolic murmur (SM) of subaortic stenosis. Note the later peaking of the systolic murmur and dimunition of $S_2$ in more severe aortic stenosis.**

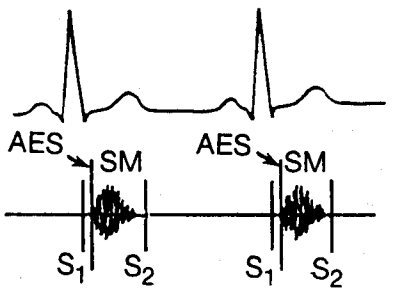


**Figure 25. Aortic ejection sound (AES) and systolic murmur (SM) in a patient with mild aortic stenosis.**

The systolic murmur of significant valvular pulmonic stenosis (with intact ventricular septum) is similar in quality to the murmur of valvular aortic stenosis (harsh or rough, medium-pitched crescendo-decrescendo). The loudness and duration of the murmur is closely related to the severity of the stenosis. The murmur is usually well localized with maximal loudness in the pulmonic area. If the murmur is loud, it is generally easily heard over the precordium. Even when the stenosis is mild, there is usually abnormal splitting of $S_2$ (persistent splitting with increased $A_2$–$P_2$ interval). Splitting may be difficult to hear owing to the decreased intensity of $P_2$ and the murmur.

Auscultatory clues in significant valvular pulmonic stenosis are as follows:

1. Harsh, medium-pitched to high-pitched, loud (grade III or greater), long ejection systolic murmur with late systolic peak (Figure 26).
2. Systolic murmur usually loudest in the pulmonic area with radiation to the right side at third intercostal space near the sternum.
3. Relatively normal $S_1$.
4. Abnormally wide splitting of $S_2$, with fixed splitting in severe stenosis.
5. $P_2$ diminished or virtually absent in severe stenosis.

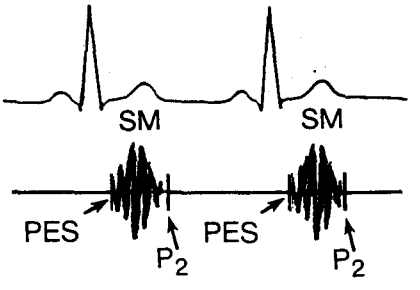


**Figure 26. The crescendo-decrescendo systolic murmur (SM) and pulmonic ejection sound (PES) of significant valvular pulmonic stenosis.**

The murmur associated with *tetralogy of Fallot* is variable and complex owing to the presence of multiple defects of variable severity. Murmurs include a right sternal border murmur associated with the ventricular septal defect and a left basilar murmur associated with the pulmonic stenosis. The pulmonic ejection murmur tends to predominate. The nature of the murmur reflects the severity of the pulmonic outflow obstruction. Mild obstruction causes a loud holosystolic murmur. Worsening obstruction causes softening and shortening of the murmur.

The systolic murmur heard in *atrial septal defect* is characteristic of systolic murmurs present in hyperkinetic or high-flow states. The murmur is crescendo-decrescendo in contour, is usually medium-pitched in quality, and is typically of short duration, ending well before the beginning of $S_2$. The murmur is generally most prominent in the pulmonic area. The murmur of atrial septal defect is not caused by blood flow across the defect; it is secondary to increased flow across the pulmonic valve and dilatation of the pulmonary artery. Small defects will not have an associated murmur.

Auscultatory clues in significant atrial septal defect are as follows:

1. Medium-pitched, short systolic murmur with characteristic diamond-shaped contour, usually soft (grade I to II), occasionally grade III in loudness.
2. Systolic murmur usually loudest at the pulmonic area (Figure 27).
3. Frequent accentuation of $S_1$ in the tricuspid valve region (owing to accentuation of the $T_1$, component of $S_1$).
4. Typical absence of $S_4$ or $S_3$.
5. Abnormally wide splitting of $S_2$, with typical "fixed" splitting during respiration.
6. $P_2$ almost always sharply accentuated.

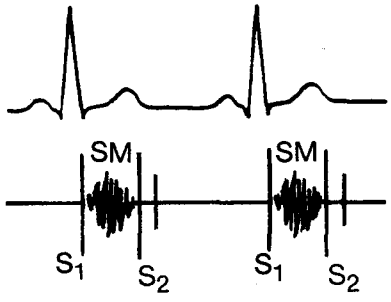


**Figure 27. Atrial septal defect. The ejection systolic murmur (SM) is followed by fixed splitting of $S_2$.**

*Murmurs associated with anemia* usually are not detected until the hemoglobin drops below 6 mg/dl. The murmur is due to turbulence caused by decreased blood viscosity. The murmur is soft (grade III or less) and occurs during early systole to mid-systole. The murmur is usually heard best at the left heart base.

*Innocent systolic murmurs* are functional (not associated with any organic heart disease). These ejection systolic murmurs may be heard in normal animals of all ages but mainly in puppies and kittens less than 6 months of age. These murmurs emanate, at least partly and probably wholly, from turbulence in the aortic root caused by forward flow across the normal right and left ventricular outflow tracts.

Auscultatory clues in innocent systolic murmurs are as follows:

1. Usually soft (grade I to II) systolic murmur of short duration in early systole (Figure 28).
2. $S_1$ and $S_2$ normal.
3. Absence of abnormal sounds (e.g., ejection sounds) or diastolic murmurs.

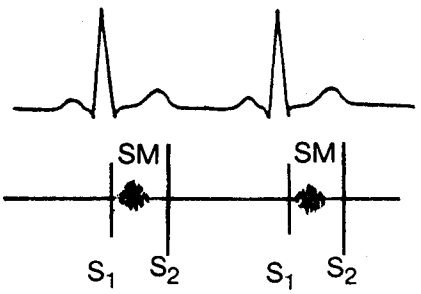


**Figure 28. Innocent low intensity ejection systolic murmur (SM) in an asymptomatic puppy.**

## HOLOSYSTOLIC OR REGURGITANT MURMURS

Holosystolic murmurs are typically characterized by (1) longer duration than ejection systolic murmurs, with murmurs beginning with $S_1$ and ending with or enveloping $S_2$; (2) a plateau configuration with relatively uniform loudness throughout systole; and (3) dominant high frequencies with a high-pitched or blowing quality to the murmur.

The typical holosystolic murmur is best initially auscultated with the diaphragm of the stethoscope. Regurgitant murmurs may be less than holosystolic and may have varying configurations, durations, and frequencies owing to the anatomic and pathophysiologic variations in the degree of regurgitation, pressure gradient during regurgitant flow, and cardiac output. Causes of holosystolic murmurs are listed in Table 16.

TABLE 16. Causes of Holosystolic Murmurs

1. Mitral regurgitation
2. Tricuspid regurgitation
3. Ventricular septal defect

*MITRAL REGURGITATION.* Regulation of blood flow across the mitral valve depends on the complex interactions of the mitral annulus, the mitral valve leaflets, the chordae tendineae, and the papillary muscles. Malfunction of any of the components of this "mitral complex" will result in an abnormal regurgitant flow of blood from the high-pressure left ventricle to the low-pressure left atrium during ventricular systole, with a resultant regurgitant murmur. The most common causes of mitral regurgitation are myxomatous degeneration of the mitral valve, ruptured chordae tendineae, and dilated cardiomyopathy.

*(1) Myxomatous degeneration of the mitral valve.* This pathologic condition is typically associated with a high-pitched mitral regurgitant murmur that is usually mid-systolic to late systolic in timing. One or more systolic or nonejection clicks (see under Mid-Systolic Clicks) may be auscultated if the valve lesion is associated with mitral valve prolapse. With progressive deterioration of the mitral valve leaflets or with superimposed ruptured chordae tendineae, a harsh, holosystolic murmur is present, frequently with late systolic accentuation. This is the most common cause of regurgitant murmurs in old, small or medium-sized dogs and occurs occasionally in old cats.

Auscultatory clues in severe degenerative mitral valve disease are as follows:

1. Harsh, loud (grade III or more) holosystolic murmur, frequently with late systolic accentuation (Figure 29).
2. Murmur typically loudest at left cardiac apex, with radiation determined by underlying pathologic condition.
3. $S_3$ and $S_4$ may be present.
4. Accentuated $P_2$ common.
5. The mid-systolic click or clicks usually obscured by the loud murmur.

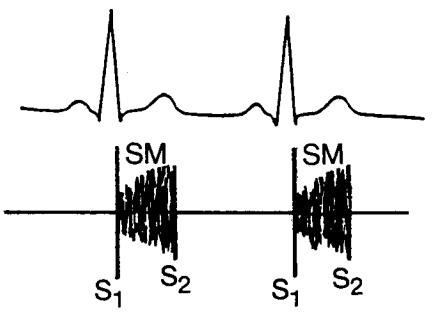


**Figure 29. The holosystolic murmur (SM) of myxomatous degeneration of the mitral valve with severe mitral valve prolapse.**

*(2) Ruptured chordae tendineae.* Such rupture may occur with acute bacterial endocarditis, myxomatous degeneration of the mitral valve, trauma, or may be idiopathic. Ruptured chordae tendineae is strongly suggested by the sudden onset of a harsh, loud (grade III or more) holosystolic (or long systolic), murmur, loudest at the apex with accompanying loud $S_3$ and $S_4$ (if in regular sinus rhythm) and accentuated $P_2$. Murmur radiation depends on which chordae tendineae are involved. In humans with posterior chordae tendineae rupture, the murmur radiates best to the base of the heart and in 50% of the cases into the neck and carotids. This murmur frequently has a diamond-shaped configuration but is holosystolic. With anterior chordae tendineae rupture in humans, murmur radiation is typically into the axilla, along the mid-thoracic vertebrae and sometimes to the top of the head. These different patterns of radiation have not been determined in dogs and cats.

Auscultatory clues in ruptured chordae tendineae are as follows:

1. Sudden onset of harsh, loud (grade III or more) holosystolic murmur, frequently enveloping $S_1$ and $S_2$ (Figure 30).
2. Murmur typically loudest at cardiac apex, radiation pattern variable depending on which chordae rupture.
3. $S_3$ and $S_4$ (when in regular sinus rhythm) may be present.
4. Accentuated $P_2$ common.

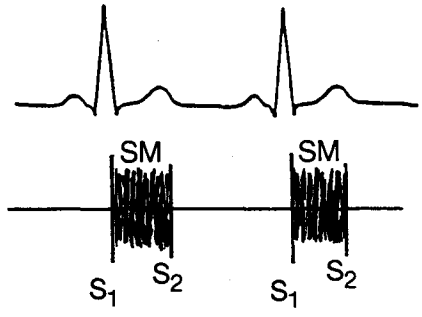


**Figure 30. The holosystolic murmur (SM) of ruptured chordae tendineae.**

*(3) Dilated cardiomyopathy.* The systolic regurgitant murmur associated with dilated cardiomyopathy is caused by stretching of the mitral valve annulus, which results in mitral valve incompetence. The valve leaflets are usually normal. The murmur is usually early systolic, plateau, or decrescendo with its point of maximal intensity (PMI) at the left apex. The murmur is usually not as loud as in patients with myxomatous degeneration of the mitral valve. Dilated or hypertrophic cardiomyopathy in cats can disrupt the normal valve anatomy, causing a regurgitant murmur.

Auscultatory clues in dilated cardiomyopathy are as follows:

1. Regurgitant systolic murmur with PMI at L5–6 ICS.
2. Murmur usually mild to moderate loudness (I–III/VI).
3. Heart sounds often decreased.
4. $S_3$ may be present.
5. Arrhythmias, especially atrial fibrillation, often present; this results in variation in loudness of heart sounds.

*TRICUSPID REGURGITATION.* The systolic murmur of significant tricuspid regurgitation is typically holosystolic and may resemble that of mitral regurgitation in timing and quality, although it is usually not louder than grade III to IV. It is heard best over the tricuspid valve area. The loudness and duration of the murmur may vary, depending on the degree or regurgitation. Tricuspid insufficiency is most commonly associated with myxomatous degeneration of the tricuspid valve. Involvement of the mitral valve is more common than the tricuspid valve and usually occurs in conjunction with tricuspid valve degeneration. It can be quite difficult to differentiate by auscultation the difference between severe mitral regurgitation with radiation to the tricuspid valve area and combined mitral and tricuspid insufficiency. Other causes of tricuspid insufficiency include pulmonary hypertension secondary to respiratory disease or heartworm disease.

Auscultatory clues in tricuspid regurgitation are as follows:

1. Holosystolic murmur, usually grade IV or less (Figure 31).
2. Murmur typically loudest in tricuspid region.
3. Right-sided $S_3$ and $S_4$ usually not present.
4. The systolic murmur frequently has characteristic respiratory variation in loudness, becoming accentuated during inspiration.
5. Persistent splitting of $S_2$, if pulmonary hypertension or heartworm disease is the cause of the valvular insufficiency.

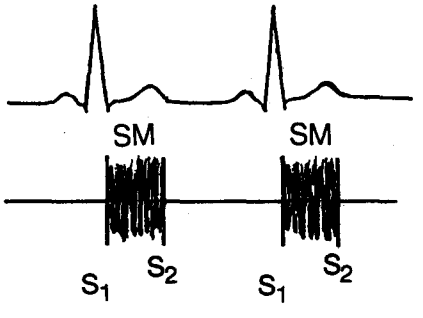


**Figure 31. The holosystolic murmur (SM) of tricuspid regurgitation.**

*VENTRICULAR SEPTAL DEFECT.* The auscultatory findings in this lesion are determined by the size of the septal defect and the pulmonary vascular resistance because these two factors primarily determine the amount and velocity of regurgitant flow from the left ventricle into the right ventricle with a resultant regurgitant murmur. With small to moderate-sized defects and normal to mildly elevated pulmonary vascular resistance (systolic pressure of 50 mm Hg or less in the pulmonary artery), the systolic murmur is typically loud (grade III to V), holosystolic, and medium to high pitched with a harsh quality, with either a "plateau" or mild diamond-shaped configuration. If the defect is small or if pulmonary hypertension accompanies a large defect, the murmur may be audible only in early systole. The murmur is usually loudest at the right cranial sternal border, but there is great individual variation in the associated heart sounds and murmur.

Auscultatory clues in moderate-sized congenital ventricular septal defect with moderate to large left-to-right shunt are as follows:

1. Harsh, loud (grade III to V) holosystolic murmur, typically "plateau" or mild diamond-shaped configuration (Figure 32).
2. Murmur typically loudest at the right cranial sternal border.
3. $S_1$ typically normal or mildly accentuated.
4. $S_2$ split usually increased, with normal respiratory variation and accentuated $P_2$.
5. Prominent left-sided $S_3$ and a mid-diastolic, low-pitched murmur (caused by high flow) may be present at the apex.
6. Occasionally soft (grade I to II) ejection systolic murmur heard in the pulmonic area, secondary to high flow and a relative pulmonic stenosis.
7. $P_2$ may increase owing to pulmonary hypertension.

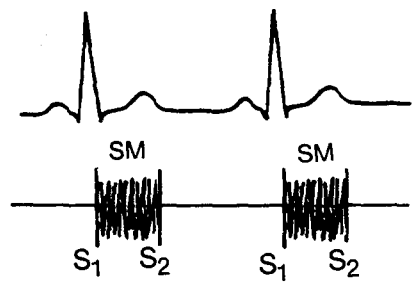


**Figure 32. The holosystolic murmur (SM) of congenital ventricular septal defect.**

## LATE SYSTOLIC MURMURS

Late systolic murmurs begin in mid-systole to late systole and end with, or envelop, the $A_2$ component of $S_2$. These murmurs are generally crescendo or diamond-shaped (crescendo-decrescendo) in configuration, are high-pitched in quality, and are heard best at the apex. One or more mid-systolic clicks are a frequent accompaniment. The most common cause of late systolic murmurs is mild to moderate mitral valve prolapse. Mitral valve prolapse is a common finding in humans but is quite rare in dogs and cats outside of the setting of myxomatous degeneration of the mitral valve and ruptured chordae tendineae.

## DIASTOLIC MURMURS

Diastolic murmurs are auscultated and recorded between $S_2$ and $S_1$ during the phases of ventricular relaxation. An auscultatable diastolic murmur strongly implies underlying organic cardiovascular disease. Diastolic murmurs may result from two main mechanisms:

1. Regurgitant flow across incompetent aortic or pulmonic semilunar valves.
2. Forward flow across stenosed mitral or tricuspid atrioventricular valves.

*Aortic regurgitation* is the most common diastolic murmur in dogs and cats. Aortic regurgitation causes diastolic murmurs that begin with or immediately after $A_2$, and are typically high frequency, high pitched, blowing in quality, and decrescendo in configuration (Figure 33). These murmurs are best auscultated with the diaphragm of the stethoscope. The most common cause of aortic regurgitation in domestic animals is bacterial endocarditis. Doppler studies have documented that aortic regurgitation often accompanies subaortic stenosis, but rarely produces an auscultatable murmur. The aortic regurgitation murmur is usually heard best in the region of the aortic valve.

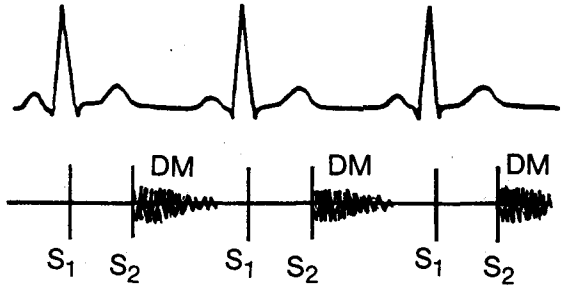


**Figure 33. Diastolic murmur (DM) in a dog with vegetative endocarditis of the aortic valve.**

There is a frequent disparity between the loudness of the aortic regurgitation murmur and the degree of regurgitation. Severe aortic regurgitation may occur with a soft, short murmur, and moderate aortic regurgitation may be present with no audible murmur (this may be most frequently seen with obese patients or patients with pulmonary emphysema, associated significant mitral valve disease, or severe left ventricular dysfunction with reduced cardiac output). A loud aortic regurgitation murmur, however, is almost always associated with significant aortic regurgitation. When valvular aortic stenosis and aortic regurgitation are combined, both the ejection systolic murmur of aortic stenosis and the diastolic murmur of aortic regurgitation are present, forming a to-and-fro murmur, and $S_2$ is often not heard (Figure 34).

Auscultatory clues in mild to moderate aortic regurgitation are as follows:

1. High-pitched, blowing decrescendo diastolic murmur starting with or immediately after $A_2$, usually grades I to III.
2. Diastolic murmur typically loudest along the aortic valve region.
3. With moderate aortic regurgitation, $S_1$ diminished.
4. $S_2$ may be narrowly split (with severe aortic regurgitation, paradoxical $S_2$ splitting may occur). $S_2$ may be accentuated (with severe aortic regurgitation or associated aortic stenosis with calcified valve, $A_2$ is diminished and often not heard).
5. Aortic ejection sound may be present.
6. Left-sided $S_3$ may be present.

The diastolic murmur of *pulmonic regurgitation* usually has the similar pitch and timing as the murmur of aortic regurgitation but is far less common. It is usually heard best in the pulmonic valve region, with an accentuated $P_2$ component of $S_2$.

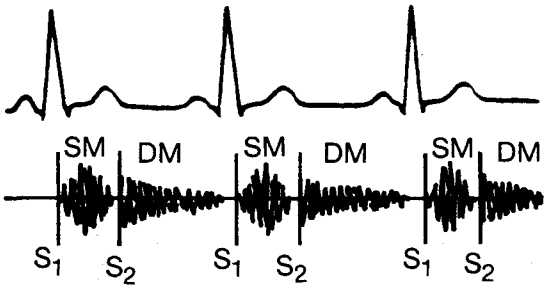


**Figure 34. The to-and-fro murmur of aortic stenosis combined with aortic regurgitation.**

*Mitral stenosis* is an extremely rare congenital defect in dogs and cats. The murmur is characteristically of low frequency, low pitched and rumbling in quality, and is therefore heard best with the bell of the stethoscope lightly applied with just enough pressure to make a skin seal. In contrast with the diastolic murmur of aortic regurgitation, there is a distinct delay in onset after $S_2$ of the diastolic murmur of mitral stenosis.

*Tricuspid stenosis* is another extremely rare congenital defect. The diastolic murmur of tricuspid stenosis is best auscultated over the tricuspid valve area. The murmur is similar to the mitral stenosis murmur but is typically softer and of shorter duration and may be accentuated during inspiration.

## CONTINUOUS MURMURS

The murmurs in this category are long murmurs that begin in systole and continue beyond the second sound into diastole.

*Patent ductus arteriosus* is a congenital lesion that is the most common cause of a pathologic or organic continuous murmur. It is the most common congenital defect in dogs and is not uncommon in cats. The murmur starts with or immediately after $S_1$ and reaches maximal loudness at or slightly before $S_2$, then is decrescendo in contour into diastole toward the next $S_1$. The classic murmur is loud (up to grade VI), is of medium pitch, and heard best at the left base (Figure 35). The diastolic component tends to be localized and the systolic component may radiate extensively. The diastolic component may be lost at the very end of diastole. It is described as a "machinery" murmur, proceeding continuously from systole into diastole. The loudness and duration of the murmur are directly related to the pressure gradient between the aorta and pulmonary artery. As pulmonary hypertension develops, the intensity of the diastolic component decreases and $S_2$ increases. If the direction of the shunt reverses, the diastolic component disappears and the systolic component at the left base becomes faint or disappears. Patent ductus arteriosus may be an isolated lesion or associated with other congenital lesions, most commonly ventricular septal defect.

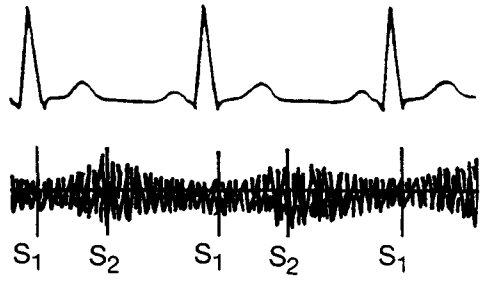


**Figure 35. Continuous murmur in a poodle with a patent ductus arteriosus.**

Auscultatory clues in patent ductus arteriosus are as follows:

1. Loud, long, "machinery" murmur, typically loudest at the left heart base.
2. Systolic component often radiates extensively.
3. Systolic regurgitant murmur often heard at the left apex.

Rare causes of organic or pathologic continuous murmurs are listed in Table 17.

TABLE 17. Other Causes (Rare) of Organic or Pathologic Continuous Murmurs

1. Aortic pulmonary window
2. Arteriovenous fistula
3. Sinus of Valsalva aneurysm rupture into the right heart
4. Coarctation of aorta (severe)
5. Coronary artery fistula
6. Pulmonary artery branch stenosis
7. Combination defects (ventricular septal defect with aortic regurgitation)

## SUMMARY

heart

Knowledge of the timing and auscultatory characteristics of heart sounds and murmurs is of paramount importance in quickly establishing a cardiac diagnosis. The characteristics of heart sounds and murmurs are visually summarized in Figure 36.

Table

urs

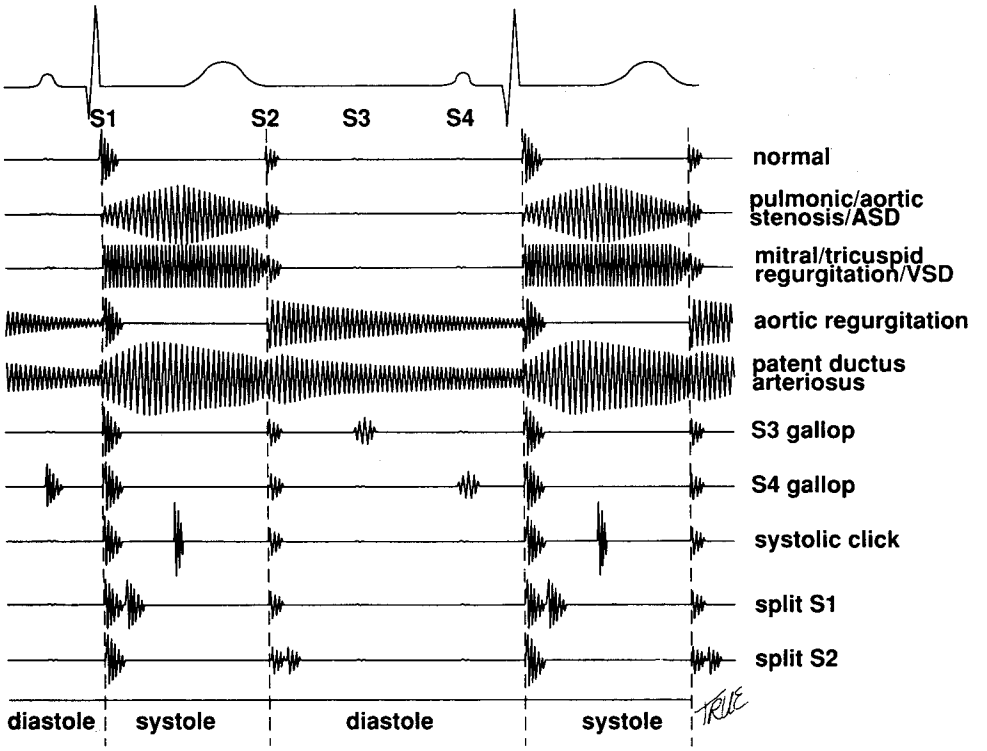


**Figure 36. Cardiac cycle with electrocardiogram and phonocardiogram schematized. Both normal and abnormal sounds are included. ASD = Atrial septal defect; VSD = ventricular septal defect. (From Atkins, C.E.: Abnormal Heart Sounds. In Small Animal Medicine. Edited by D.G. Allen. Philadelphia, J.B. Lippincott, 1991, p. 198.)**

## ANSWERS TO PRETEST 2

1. IV or greater
2. Right cranial sternal border, left apex, or both
3. Pulmonic stenosis, aortic stenosis
4. False
5. True
6. False
7. True
8. Accentuated
9. True
10. True

# POST-TEST 2

## PART A

*DIRECTIONS.* Part A consists of 10 questions. After determining the correct answers, fill in the appropriate blanks.

1. The more severe the aortic stenosis, the _____ the peak loudness of the murmur occurs.
2. List three breeds of dogs that frequently get pulmonic stenosis. _____, _____, _____.
3. The murmur of tetralogy of Fallot consists of an ejection murmur and a regurgitant murmur. True or False _____.
4. A "machinery" murmur may be heard in _____.
5. The murmur of an atrial septal defect is caused by shunting of blood between the left atrium and the right atrium. True or False _____.
6. Innocent murmurs are more commonly auscultated in young animals (<6 months) than adults. True or False _____.
7. A grade III murmur may be heard even with the stethoscope off the chest. True or False _____.
8. A diamond-shaped systolic murmur is characteristic of tricuspid regurgitation. True or False _____.
9. The murmur in congenital subaortic stenosis is always present from birth. True or False _____.
10. The murmur of mitral regurgitation is heard best at the right apex. True or False _____.

## PART B

*DIRECTIONS.* Part B consists of five unknowns presented on your cassette tape, side 2. After determining the correct answers, fill in the appropriate blanks. Pay close attention to the location and timing of the murmurs. Because you are not examining the patient, the location and, where appropriate, the timing, are provided.

1. Aortic area—systolic. _____.
2. Pulmonic area. _____.
3. Pulmonic area. _____.
4. Apex—systolic _____.
5. Aortic area—diastole. _____.

# ANSWERS TO POST-TEST 2

## PART A

1. Later
2. Choose from Beagle, Samoyed, Bulldog, Chihuahua, Fox Terrier, and Miniature Schnauzer
3. True
4. Patent ductus arteriosus
5. False
6. True
7. False
8. False
9. False
10. False

## PART B

1. Ejection systolic murmur of aortic stenosis
2. Continuous or "machinery" murmur of patent ductus arteriosus
3. Systolic murmur and fixed splitting of $S_2$ in atrial septal defect
4. Holosystolic murmur of myxomatous degeneration of the mitral valve
5. Aortic insufficiency in patient with endocarditis

# ARRHYTHMIAS

## OBJECTIVES (SECTION 3)

Upon completion of this program, you should be able to:

1. Recognize the limitations of auscultation in the definitive diagnosis of arrhythmias.
2. Characterize the auscultatory findings of atrial fibrillation.
3. Integrate physical findings with auscultatory findings to characterize the arrhythmia better.
4. Differentiate normal from abnormal rhythms in different species.
5. Appreciate the difference between a gallop rhythm and an arrhythmia.

## PRETEST 3

*DIRECTIONS.* This pretest consists of five questions. After determining the correct answers, fill in the appropriate blanks.

1. Atrial fibrillation is characterized by a rapid irregular rhythm with variable loudness of the heart sounds. True or False _____.
2. Paroxysmal atrial tachycardia and paroxysmal ventricular tachycardia can be reliably differentiated on auscultation. True or False _____.
3. Respiratory sinus arrhythmia is characterized by an _____ in the heart rate on inspiration and an _____ of the heart rate on expiration.
4. An $S_4$ associated with atrial contraction sometimes is heard in patients with complete heart block. True or False _____.
5. Sinus arrhythmia is generally a pathologic finding in _____ (species) and a normal variant in _____ (species).

Answers on page 62.

## AUSCULTATION OF SELECTED ARRHYTHMIAS

Arrhythmias are most accurately diagnosed by electrocardiography. The first clue that an animal has an arrhythmia, however, is found on auscultation. Some arrhythmias have distinctive auscultatory findings, whereas others are indistinguishable on auscultation. Any time an irregularity in heart rate or rhythm is detected on auscultation, an ECG should be performed. A few selected arrhythmias are discussed here.

Sinus rhythm is a very regular rhythm that does not vary with respiration. Sinus rhythm is normal in dogs and cats.

Sinus arrhythmia is an irregular rhythm, usually associated with a normal heart rate. The irregularity is generally regular and associated with the phases of respiration. On inspiration, there is a drop in vagal tone that results in an increase in the heart rate. The degree of irregularity can be increased by conditions that increase vagal tone. These would include respiratory and gastrointestinal disturbances. A respiratory sinus arrhythmia is a normal variant in dogs but is abnormal in cats. It is most frequently associated with asthma in cats.

Auscultatory and physical findings in sinus arrhythmia include the following:

1. Normal heart rate.
2. Regularly irregular rhythm.
3. Heart rate varies with respiration, increasing on inspiration.
4. Pulse quality may vary slightly, but there are no pulse deficits.

Associated clinical conditions are as follows:

1. Normal variant in dogs.
2. Accentuated by conditions that increase vagal tone (respiratory and gastrointestinal disorders) in dogs.
3. Bronchial asthma in cats.

Atrial fibrillation is characterized by a rapid irregularly irregular rhythm. The loudness of the heart sounds is also variable from beat to beat.

Auscultatory and physical findings in atrial fibrillation (Figure 37) include the following:

1. Rapid heart rate.
2. Irregularly irregular rhythm.
3. Irregularity not associated with respiration.
4. Pulse quality usually decreased, irregular in fullness with frequent pulse deficits.
5. Difficult to correlate heart beats with pulses.

Associated clinical conditions are as follows:

1. Dilated cardiomyopathy in dogs.
2. Hypertrophic cardiomyopathy in cats.
3. Severe mitral regurgitation secondary to valvular disease in dogs.

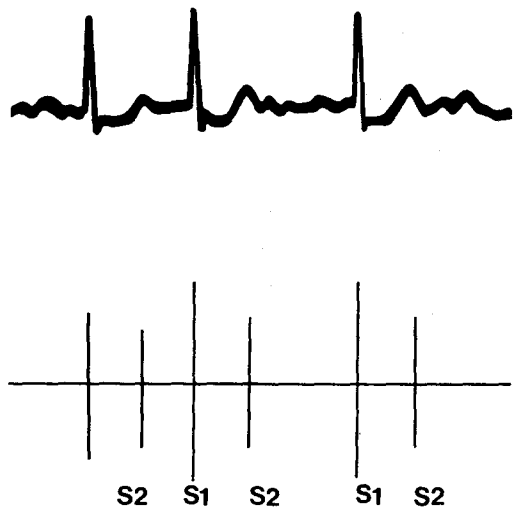


**Figure 37. Atrial fibrillation in a Doberman Pinscher. Note the irregularity in the rhythm and the variability in the intensity of the heart sounds.**

Auscultatory findings with complete heart block may also be chaotic, but the heart rate is slow to normal. $S_1$ and $S_2$ occur at regular intervals and usually at a slow rate. There may be some variation in the loudness of the first and second heart sounds owing to variable ventricular filling. $S_4$ may be audible in association with atrial contraction. The $S_4$–$S_4$ interval may be regular or irregular depending on whether the underlying rhythm is a sinus rhythm or sinus arrhythmia. There is no association between the $S_1$–$S_2$ and $S_4$.

Auscultatory and physical findings in complete heart block (Figure 38) include the following:

1. Rhythm may be slow and regular if $S_4$ is not audible or irregular and chaotic if $S_4$ is audible.
2. Pulses regular even if the rhythm sounds chaotic. Pulses follow $S_1$ and precede $S_2$. Atrial contraction ($S_4$) does not produce a femoral pulse.
3. Jugular pulses often visible when atrial contraction occurs during ventricular systole. As the AV valves are closed during ventricular systole, blood is forced retrograde up the jugular veins.

Associated clinical conditions are as follows:

1. AV nodal fibrosis or degeneration in geriatric animals.
2. Cardiomyopathy, especially in cats.
3. Neoplastic infiltration into AV node.
4. Endocarditis or myocarditis (Lyme disease).

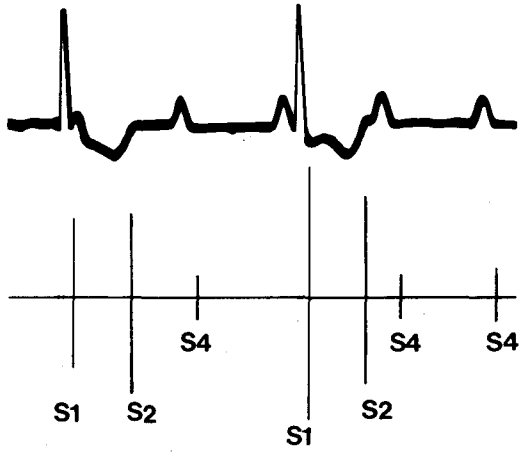


**Figure 38. Complete atrioventricular block in a 12-year-old Cocker Spaniel. When a P wave occurs before the QRS complex (2nd complex), the first heart sound is accentuated. Note an $S_4$ may be associated with the nonconducted P waves.**

Ventricular premature complexes (VPC) should always be suspected when a heart beat occurs prematurely and is associated with a pulse deficit.

Auscultatory and physical findings with ventricular premature complexes (Figure 39) include the following:

1. A regular or regularly irregular rhythm is interrupted by a premature beat.
2. The premature beat often fails to produce a palpable femoral pulse (pulse deficit). The more premature the beat, the weaker the pulse. A late diastolic VPC may not produce a perceptible change in pulse quality.
3. Heart sounds may be split owing to asynchronous depolarization of the ventricles by the VPC.

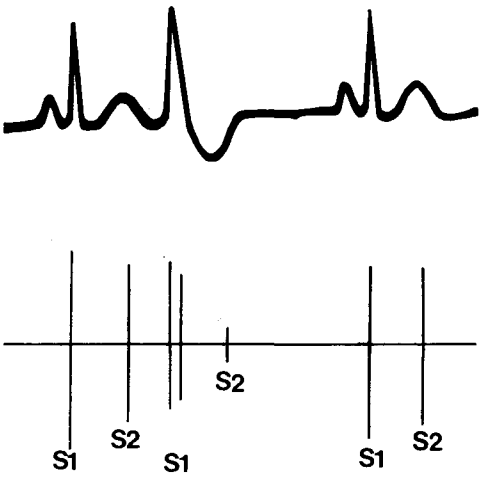


**Figure 39. Ventricular premature complex (2nd complex) in a dog. Note the split first heart sound and softer $S_2$.**

Common associated clinical conditions are as follows:

1. Cardiomyopathy in dogs and cats.
2. Hyperthyroidism in cats.
3. Traumatic myocarditis in dogs.
4. Systemic diseases (gastric torsion, pancreatitis and hemangiosarcoma) in dogs.

Note: VPCs and atrial premature complexes (APC) cannot reliably be differentiated on the basis of auscultation. Both occur prematurely and can cause pulse deficits. APCs maintain atrial and ventricular synchrony and therefore will have a stronger pulse for any given degree of prematurity. This is a useful distinguishing finding only if the patient has APCs and VPCs. VPCs may produce a jugular pulse, APCs do not.

Paroxysmal ventricular and atrial tachycardia (PVT, PAT) are characterized by bursts of tachycardia that begin and end abruptly. Patients may have periods of sinus tachycardia that can be confused for PAT or PVT. Tachycardic episodes associated with sinus tachycardia, however, tend to speed up and slow down gradually rather than change abruptly. PAT and PVT cannot accurately be differentiated by auscultation and require ECG confirmation.

Auscultatory and physical findings in PAT and PVT (Figure 40) include the following:

1. Bursts of tachycardia (heart rate up to 400) that begin and end abruptly.
2. Patients may also have isolated premature beats (APC or VPC).
3. Pulses become weak or absent during tachycardic episodes.

Common conditions associated with paroxysmal atrial tachycardia are as follows:

1. Atrial distention secondary to AV valve disease.
2. Ventricular pre-excitation.
3. Cardiomyopathy.

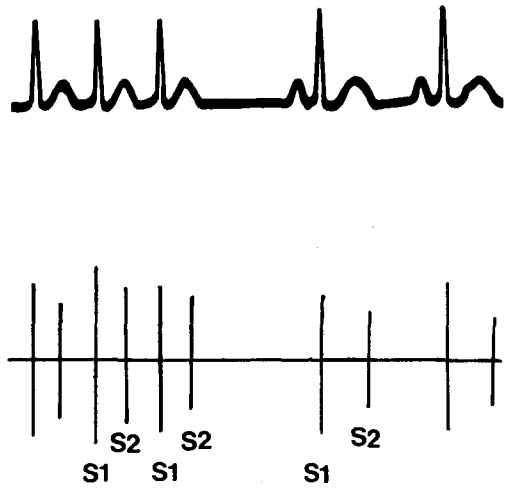


**Figure 40. Paroxysmal atrial tachycardia (first three complexes) in a 10-year-old Poodle.**

Sustained sinus tachycardia, ventricular tachycardia, or atrial tachycardia cannot be reliably differentiated based on auscultation. Any patient with sustained tachycardia should have an ECG performed.

Gallop rhythms as discussed previously are caused by extra heart sounds ($S_3$ & $S_4$). These extra sounds are sometimes confused for an arrhythmia. These sounds are caused by changes in mechanical energy rather than electrical energy, and the ECG rhythm is unaffected.

## ANSWERS TO PRETEST 3

1. True
2. False
3. Increase, decrease
4. True
5. Cats, dogs

# POST-TEST 3

## PART A

*DIRECTIONS.* Part A consist of 10 questions. After determining the correct answers, fill in the appropriate blanks.

1. Most arrhythmias can be diagnosed on auscultation. True or False _____.
2. A jugular pulse is more likely to be seen with atrial or ventricular premature complexes? _____.
3. The more premature the ectopic beat occurs, the louder the associated heart sounds. True or False _____.
4. Auscultating the heart during a vagal maneuver can help differentiate sinus, atrial, and ventricular tachycardia. True or False _____.
5. A gallop rhythm is a form of arrhythmias that can be detected on auscultation and on an ECG. True or False _____.

## PART B

*DIRECTIONS.* Part B consists of five unknowns presented on your cassette tape, side 2. After determining the correct answers, fill in the appropriate blanks. Some cases have more than one possible answer.

1. Doberman Pinscher with dilated cardiomyopathy _____.
2. Coughing dog with bronchitis _____.
3. Geriatric terrier with history of syncope _____.
4. German Shepherd that was recently hit by a car _____.
5. Middle-aged King Charles Spaniel with coughing _____.

# ANSWERS TO POST-TEST 3

## PART A

1. False
2. Ventricular
3. False
4. True
5. False

## PART B

1. Atrial fibrillation
2. Sinus arryhthmia
3. Paroxysmal atrial tachycardia
4. Ventricular tachycardia. Atrial tachycardia would also have to be considered based on auscultation. The history supports traumatic myocarditis, and therefore ventricular tachycardia would be more likely. Split heart sounds also support ventricular tachycardia. An ECG is needed for a definitive diagnosis.
5. Ventricular premature complexes. Atrial premature complexes would also have to be considered based on auscultation. An ECG was performed to confirm the diagnosis. Note that the VPCs in this patient do not have audible splitting of the heart sounds. A systolic murmur is present.